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ASIAN INFLUENZA IN LOUISIANA IN SUMMER OF 1957 *

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During April of 1957, many persons in Hong Kong, China, developed the typical clinical picture of influenza. The disease apparently originated in the northern section of China and then spread rapidly throughout the Orient with a reported attack rate of 10 to 20 per cent. The causative agent was identified in May at the Walter Reed Army Institute of Research as a new variant of type A influenza virus, termed Far East influenza A, and popularly designated the Asian strain. Serologic studies in United States military personnel indicated a lack of prior experience with the agent or related strains.[1]

The first confirmed case of Asian influenza in the continental United States occurred in June among U. S. Navy personnel in Newport, Rhode Island, followed by other case reports from California.[2] It was soon apparent that Louisiana was experiencing an outbreak of epidemic proportions.

In retrospect, the first occurrence of influenza in Louisiana was probably at a boy scout camp near Slidell, Louisiana, during the last week of June. A clinical diagnosis of streptococcal sore throat made on 26 campers was not substantiated by throat cultures. Throat garglings and paired sera were not obtained for virus studies.

On July 14, 45 out of 60 persons attending a church camp near Pollock, Louisiana, became ill with an influenza-like illness. The Asian strain of influenza A virus was isolated from the throat washings of 2 of the sick individuals. Paired sera from 3 others showed a fourfold or greater rise in titre of hemagglutination-inhibition antibody to Asian strain influenza A virus. This outbreak was probably originated by a person who had been exposed to a sick member of a Bossier City, Louisiana, band which had just returned from California.

In mid-July 37 scouts suffered an influenza-like illness while en route by bus to the Boy Scout Jamboree in Pennsylvania. Several had attended the scout camp at Slidell, Louisiana in June. An outbreak involving 100 negro males of the 950 employed in two fish processing factories in Plaquemine Parish, Louisiana, also occurred in the early part of August.

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The Asian strain of virus was isolated from the throat washings of 1 of these men.

The most widespread outbreak of influenza in Louisiana occurred in Tangipahoa Parish (population 60,000), during the first part of August. A state charity hospital (Lallie Kemp Hospital) serving the area estimated at least 8,000 persons were sick during the first week of August. Later in the month, physicians reported 3,766 additional cases. Asian strain, influenza virus was isolated from 2 of these and 16 others had a fourfold or greater rise in hemagglutination-inhibition antibody titre to Asian strain virus in convalescent sera. The mechanism for the localization of the first large epidemic in Louisiana is not known. Tangipahoa Parish is one of the few places in the United States where public schools are in session during the summer months. The regular vacation period is in the spring in order that children may help with the annual strawberry harvest. The close contact in classes and school buses may have been responsible for the development and spread of the epidemic in this area in the summer. Influenza appeared in the New Orleans area soon afterwards.

This report describes 5 cases of Asian influenza which were studied on the Tulane Medical Service of the Charity Hospital of Louisiana and the New Orleans Veterans Administration Hospital.

MATERIALS AND METHODS

Fifteen patients with a typical clinical syndrome of influenza were admitted during a three day period, August 8 to 11, for detailed study. During this period 11 percent of the 1,518 negro patients who visited the Charity Hospital Admitting Room were diagnosed clinically as having influenza. Concurrently, 7 white patients of 450 (1.5 per cent) were considered to have influenza. The clinical diagnosis of influenza due to the Asian strain of virus was confirmed in the 15 hospitalized patients by virus isolation from throat washings in the chick embryo or monkey kidney cultures [3] or by a fourfold or greater rise in hemagglutination-inhibition antibody titre in convalescent sera.

CASE REPORTS

Patient No. 1—O. B. This 27 year old healthy colored male was awakened about 4 a. m. by nocturia which had not existed previously. Six hours later he began to have a right-sided headache and right lumbar pain followed by a chill and fever. There was no cough, coryza, or history of recent travel or exposure to ill persons.

Physical examination: On admission to Charity Hospital on August 9, 1957, he appeared to be acutely ill. Temperature was 103°; pulse 84; respiration 20; and blood pressure 130/90. There was bilateral injection of the conjunctivae, but there were no inflammatory changes in the nose or pharynx. The neck was supple, the eye grounds were normal and no lymphadenopathy or skin eruption was apparent. A few crepitant rales were present at both bases which cleared with deep breathing. The heart and abdomen were normal. Rectal, prostatic, and neurological examinations also were normal.

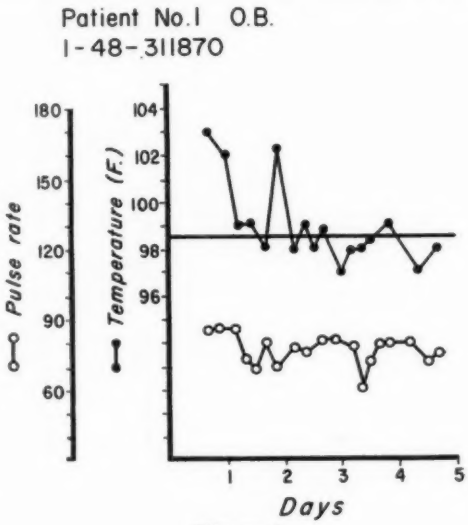

Figure 1

Laboratory Data: WBC 4,700, neutrophiles 70%, lymphocytes 28%, eosinophiles 2%, hematocrit 49%. Urinalysis and transaminase were normal; urine and blood cultures were negative; throat culture demonstrated no obvious pathogen or predominant organism. Chest x-ray was normal. The ECG showed inverted T waves in standard leads II and III.

Course in hospital: The patient received symptomatic treatment only. After a brief elevation in temperature on the second day, he became afebrile with disappearance of all symptoms. At the height of his fever a short apical systolic murmur was audible which disappeared on the following day.

The urinary symptoms and mild diastolic hypertension were also present only during the period of fever, and he was entirely asymptomatic at time of discharge. The diagnosis of influenza, Asian strain was made by isolation of virus from throat washings obtained at time of admission. Follow-up examination in two weeks was entirely negative, including the ECG, although the patient complained of leg pains with prolonged walking.

Patient No. 2—W. T., a 25 year old colored male, previously in good health, had sudden onset of fever and generalized weakness while at work. At home he had a severe rigor lasting about five minutes and a frontal headache developed which was intensified with the slightest movement of the head. There was an intense retro-orbital discomfort with eye movement, a nonproductive cough, and dryness of nose with occasional sneezing. He had retrosternal ache after coughing and weakness of the extremities but no definite muscle aches. There were no gastrointestinal or urinary symptoms and no history of exposure to any persons with the same type of illness. He was admitted to Charity Hospital on August 9, 1957.

Physical examination: Examination revealed a well developed, well nourished male, appearing acutely ill sweating profusely, and lying quietly in bed. Blood pressure was 130/70, pulse 100, temperature 104°, and respiration 27. The conjunctival, nasal, and pharyngeal membranes were injected. Several small petechiae were noted on the soft palate. There were a few transient crepitant rales heard at both lung bases. The heart sounds were normal and there was no lymphadenopathy. The remainder of the physical examination was normal.

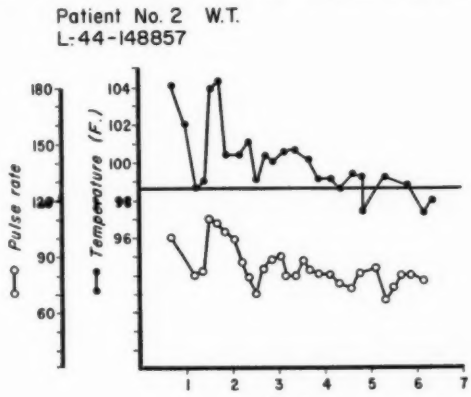

Figure 2

Laboratory Data: WBC 10,300, neutrophiles 76%, lymphocytes 22%, monocytes 2%, hematocrit 41%. Urinalysis and transaminase were normal; urine and blood cultures were negative; throat culture demonstrated no obvious pathogen or predominant organism. Chest x-ray and ECG were normal.

Course in hospital: Symptomatic care was administered consisting of bed rest, fluids and codeine. He remained febrile for four days during which time the persistent dry cough greatly aggravated the retrosternal ache. His vital signs remained stable, however. He was asymptomatic when discharged on the sixth day. Throat washings taken on admission were positive for influenza A, Asian strain.

Patient No. 3—L. L. This 40 year old colored nurse's aid was exposed to her 3 year old child who had fever 103.2°, dry cough and conjunctival injection. Two days later, one day before admission, there was a sudden onset of chills, fever, and diffuse pains in the anterior region of the chest, aggravated by breathing. Temperature was 104° and myalgia, arthralgia, and headache were present. The patient then developed anorexia, vomited once, and had mild continuous nausea but no diarrhea. Lacrimation, mild sore throat, and generalized skeletal muscle weakness were present. Soon after the onset of symptoms she developed a dry cough which greatly aggravated the chest pain. She was admitted to Charity Hospital on August 8, 1957.

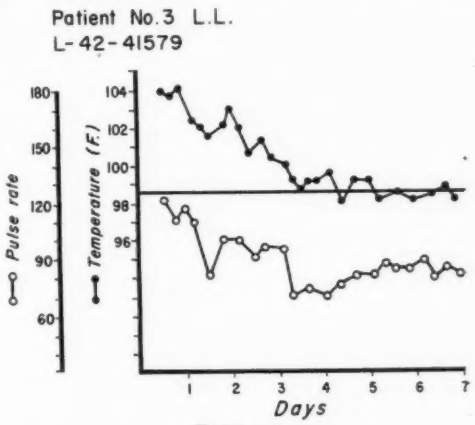

Figure 3

Physical examination: The patient was a well-developed, well-nourished female who appeared acutely ill and "toxic". She complained of chest pain, was tachypneic, and had frequent episodes of coughing. Blood pressure was 130/70, pulse 120, temperature 104°, and respiration 28. She was sweating profusely, had a flushed face but no cutaneous erruption. The anterior cervical lymph nodes were tender but not significantly enlarged. All other lymph nodes were normal. The conjunctivae, uvula, and pharynx were injected. Several petechiae were seen on the soft palate. The chest expanded symmetrically with shallow excursions, and was hyperresonant to percussion because the

patient restricted the extent of expiration. A few crepitant rales were heard at both bases posteriorly. The heart, and abdomen were normal. There were no localizing neurological findings, although the tendon reflexes were more active than usual. Generalized skeletal muscle weakness and tenderness were significant findings. The patient appeared mentally normal but was restless.

Laboratory Data: WBC 3,400, neutrophiles 76%, lymphocytes 23%, monocytes 1%, hematocrit 37%, sedimentation rate 44 mm. Urinalysis and transaminase were normal; urine and blood cultures were negative; throat culture demonstrated no obvious pathogen or predominant organism. The chest x-ray was normal. ECG showed low T_1 and T in V_4, V_5 and V_6 to be diaphasic or inverted.

Course in hospital: The patient was very uncomfortable due to fever, and substernal chest pain related to respiration. She was treated symptomatically with codeine and sponge baths and became afebrile in five days. During the febrile period blood pressure was normal, whereas the pulse and respirations were accelerated. The tachycardia was out of proportion to the fever and was accentuated by minimal exertion such as sitting upright in bed. This occurred only during the acute phase of the illness. An electrocardiogram recorded during the height of the fever showed T wave abnormalities which improved after recovery. Throat washings obtained on admission were positive for Asian strain of influenza A. She was discharged on the seventh day, asymptomatic except for mild cough on exertion which persisted for another week.

Patient No. 4—E. B., a 31 year old colored male was admitted to the New Orleans Veterans Administration Hospital on August 10, 1957, because of sudden onset of chills, fever, frontal headache,

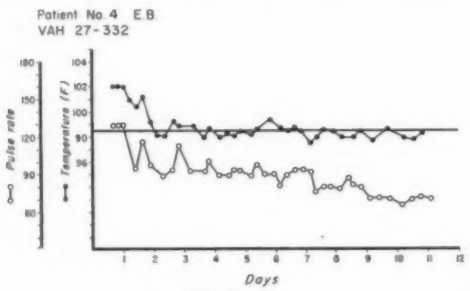

Figure 4

malaise, and weakness of lower extremities of one day's duration. There had been a frontal headache five days prior to admission with mild dysuria for the previous three days. On the day of admission he developed a dry cough, sensation of scratchiness in the throat, but no nausea, vomiting, hematuria, or urinary frequency. The patient was an operating room assistant but had not known of specific exposure to persons with febrile illness.

Physical examination: The patient appeared acutely ill and complained of diffuse muscular aches. Temperature was 102.2°, pulse 120, respiration 22, blood pressure 130/90. There were a few slightly enlarged posterior cervical lymph nodes. The conjunctival and pharyngeal membranes were injected and the nasal turbinates were edematous and mildly inflamed. The lungs were clear and the heart was normal except for a sinus tachycardia. The abdomen was normal but there was mild pain to heavy palpation in the right lumbar region. The prostate was slightly tender.

Laboratory Data: WBC 4,300, neutrophiles 43%, lymphocites 50%, and eosinophiles 7%, hematocrit 50%, sedimentation rate 27 mm. Urinalysis and transaminase were normal; urine and blood cultures were negative; throat culture demonstrated no obvious pathogen or predominant organism. Chest x-ray and ECG were normal. The diagnosis was confirmed by a fourfold rise in titre of hemagglutination-inhibition antibody to Asian strain influenza A virus in convalescent serum.

Course in hospital: The patient responded to symptomatic treatment with codeine and atropine, became afebrile in three days and was asymptomatic on discharge.

Patient No. 5—C. O., a 23 year old colored male developed a frontal headache, chills, and fever on the day of admission to New Orleans Veterans Administration Hospital, August 8, 1957. There had been anorexia, malaise, myalgia, and fever for one week. He had had protracted symptoms of an upper respiratory infection for several months,

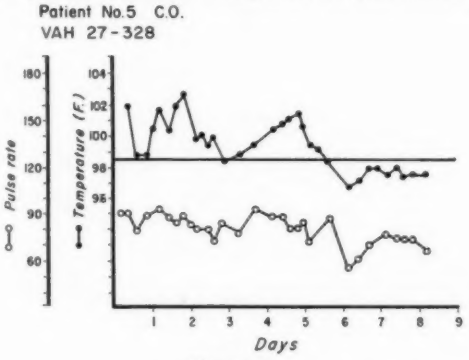

Figure 5

with nasal congestion, rhinorrhea and cough, all of which had become more severe at the time of admission. He had noted a slight darkening of urine and his cough became productive of clear mucus. There was no history of exposure to ill persons.

Physical examination: Blood pressure was 98/60, pulse 90, temperature 102°. The patient appeared acutely ill. The conjunctival, nasal, and pharyngeal membranes were mildly inflamed but without any purulent exudate. The paranasal sinuses were

not tender. Small posterior left cervical lymph nodes were palpable. Heart, lungs, abdomen, rectum and prostate were clinically normal. There was bilateral lumbar tenderness. Neurological examination was normal.

Laboratory Data: WBC 9,400, neutrophiles 74%, lymphocytes 20%, monocytes 4%, eosinophiles 2%, hematocrit 49%, sedimentation rate 23 mm. Urinalysis and transaminase were normal; urine and blood cultures were negative; throat culture demonstrated no obvious pathogen or predominant organism. Chest x-ray and ECG were normal.

Course in hospital: Therapy was symptomatic. The temperature began to decline on the third day only to rise again to the original level on the fifth day after admission. The cause for this secondary rise in temperature was not apparent and he was asymptomatic except for mild nausea and backache. The leukocyte count at this time was 5,800, neutrophiles 57%, lymphocytes 42%, monocytes 1%, hematocrit 49% and sedimentation rate 14 mm. The temperature became normal on the sixth day and the patient was discharged asymptomatic after eight days in the hospital. Influenza A virus, Asian strain was isolated from throat washings collected on the day of admission.

THE CLINICAL SYNDROME

These patients had an abrupt onset of their illness with fever, watery nasal discharge, burning sensation of the conjunctivae, soreness in the pharynx, headache, fatigability and an increase in effort required for physical and mental activity. This was followed rapidly by chilly sensations or rigors, higher fever, cough, myalgia, and pain in the eyeballs. The myalgia involved the back and legs and was severe, requiring aspirin and codeine for relief in 2 of the 5 patients. Weakness, chest pain, nausea, and photophobia were noted, and impairment of mental function with confusion and apprehension were prominent manifestations during the febrile period. Three male patients had dysuria and frequency without urinary tract infection.

All patients appeared seriously and acutely ill at the height of their fever, 102° to 104°, which usually remained at the maximal level for one or two days, and then subsided, returning to normal in about five days. Two of the 5 patients had a diphasic temperature curve, (Figures 1 and 2). Several petechiae were seen on the soft palate of 2 patients. The pharyngeal, nasal, and conjunctival membranes were injected. The face was flushed and the skin was hot and dry. Three of the 5 patients had cervical lymphadenopathy, 1 anterior and 2 posterior. The lung fields contained transient crepitant rales. The liver and spleen were not palpable. The muscles of the extremities and back were tender. Three patients had leukopenia and 2 had slight leukocytosis. Two patients had abnormal T waves in the electrocardiogram which improved with recovery.

The clinical course was typical of moderately severe influenza with fairly prompt recovery from the toxic manifestations. One patient had diffuse pains in the legs for one week after discharge and one had a nonproductive cough on exertion for several days after leaving the hospital. Complete recovery was rather slow with weakness, fatigability, and sluggishness of mental functions as the chief residual complaints.

Therapy consisted of bed rest, bland and soft diet, aspirin for fever with codeine added for headache and myalgia. Good nursing and close observation for complications were maintained until discharge. None of the 5 patients developed any complications and no antibiotic therapy was employed.

DISCUSSION

These 5 patients presented the classical manifestations of influenza so well described in the standard textbooks of medicine. All of the patients studied displayed maximal degree of illness during the first twenty-four to forty-eight hours. They were obviously extremely ill initially and some patients thought they might die. Impairment of mental function existed to some degree in the more severely ill patients.

Five patients in this area have died, 4 of fulminating pneumonia and 1 with pericarditis. Each patient had clinical manifestations compatable with influenza with cardiopulmonary involvement resulting in the terminal state. The nasal secretions sera, and tissues of these cases are

under study, and it is believed that death resulted from a bacterial complication of influenza. Serologic evidence strongly suggestive of previous infection by Asian strain influenza virus has been obtained in the pre-mortem sera of 3 of these patients; the diagnosis has been confirmed in the wife of one of these patients. Also, a diagnosis was made clinically and confirmed by rise in titre of hemagglutination-inhibition antibody in the mother of an infant who was dead on arrival at the Lallie Kemp Hospital during the Tangipahoa Parish epidemic.

Careful and prompt bacteriologic study of secretions by means of a stained smear and cultures should be obtained in any patient who presents evidence of a complication or a variation in the course of his illness. Antibiotic therapy of a bacterial complication should be started early, the duration and type as dictated by the clinical course and results of the stained smear until the culture and antibiotic sensitivity report are obtained. The fulminating nature of these complications would indicate the need for large doses of antibiotics

The greater frequency and severity of the disease in the negro population exemplified by these 5 proven cases and by the high incidence in the Charity Hospital Admitting Room has not been explained.

It would appear from present experiences in Louisiana and past experiences with influenza that the epidemic will spread following opening of schools and the advent of winter, unless an adequate vaccine can be administered to the population in sufficient time. A nationwide epidemic would greatly overburden medical facilities in addition to causing considerable economic losses. Physicians must acquaint themselves with the clinical manifestation of influenza and complications of this illness. Facilities for proper virologic and bacteriologic studies should be available to assist with the special and more complex problems of bacterial complications.

SUMMARY

A study of 5 cases of influenza due to the Asian strain of influenza A virus revealed the disease to be typical of moderately severe classical influenza. In this study the incidence and severity appeared greater in negro patients. All 5 patients recovered quickly from the more acute manifestations but weakness, fatigability, and skeletal muscle soreness persisted for several days.

Five deaths among other suspected cases of influenza yet under study, have apparently resulted from fulminating bacterial complications. Prompt treatment of such complications with large doses of antibiotics is stressed. Uncomplicated cases under careful surveillance require only bed rest, a soft bland diet, and symptomatic therapy.

A continued spread of influenza of nationwide epidemic proportions is anticipated with the opening of schools and the onset of winter.

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PALLIATION IN CANCER THERAPY *

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ROBERT B. CLIFTON, M. D. †
SHREVEPORT

"To cure sometimes, to relieve often, to comfort always."

It is a fact, well known to us all, that with the conquest of infectious diseases, with increasing longevity, the treatment of cancer has assumed correspondingly greater significance. "Progress in dealing with the cancer problem must not be measured

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solely by the cure rate, but also by the advances in palliative therapy." [11]

This is an important concept. Naturally, we seek a cure, but there are many other chronic diseases which, though not necessarily fatal, are incurable and in which palliative therapy is accepted as the method of management. Palliation is a principle as old as mankind. Certainly the earliest medical records indicate efforts to alleviate the effect of diseases, cancer included. It is a principle we all accept and use without, frequently, clearly defining our objectives or appreciating our accomplishments.

The size of the problem is immediately evident when we recall that, grouping all types of cancer together, the overall cure rate is only 25 per cent. That means that 75 per cent of cancer cases will require some type of palliation in the course of their illnesses. And studies [29] have indicated that in these cases needing palliation, in approximately 75 per cent effective and significant palliation can be accomplished. This may vary from simply relief of pain in the terminal phases to definite prolongation of life.

We do not consider mere prolongation of life per se as palliation. To have a patient in the terminal phases of malignancy, and to keep him alive a few days by the use of heroic measures of stimulation, oxygen, infusions, is not palliation in its true sense. Palliation implies keeping a patient comfortable, possibly productive, and certainly responsive to his environment rather than vegetating.

"Postoperative duration of life is not the proper measure of palliation in the care of incurable cancer. Relief of symptoms, freedom from distress, and a sense of well-being far outweigh the importance of the duration of living." [28]

We may carry out what we, at the time, consider and hope to be a curative procedure, e.g., a radical mastectomy in a case that is by all evidences clinically curable. Yet if that patient dies ten years later from the effects of that cancer, we are forced to the realization that we actually carried out a palliative procedure which undoubtedly prolonged the patient's life. We cannot forget that curative surgery does not always cure, but sometimes only palliates, and occasionally what is considered to be a palliative procedure, sometimes effects a cure. None can deny that unpredictable and sometimes unaccountable benefits accrue.

Our prime purposes in palliation are to relieve distress and to rehabilitate to a comfortable and useful life for as long as possible. After all, palliation, in effect, approximates our primary functions as physicians: It is not our job to drive death from the face of the earth, but to prolong life and to make that life more comfortable. These then, are the basic premises of palliative therapy: To prolong life and to relieve symptoms. Or in terminal states, as beautifully expressed by Axel Munthe in "The Story of San Michele": Our mission is "to help those to die we cannot help to live." This, I might say, parenthetically, is not to be considered an argument for euthanasia. In all cancer therapy, we reassure according to our hopes, and treat according to our fears.

In cancer therapy, the aims of palliation are met in three ways:

1. By causing temporary regression or cessation of cancer growth.
2. By relieving lethal complications such as intestinal obstruction or disabling complications as pathologic fractures.
3. By physical and mental relief of pain, anorexia, nausea, etc., and supportive treatment to improve the general welfare of the patient.

Disregarding the obvious measures of transfusions, narcotics, nutrition, etc., we find that there are four modalities of therapy useful in cancer palliation:

1. Surgery.
2. Radiation — cytotoxic agents acting by virtue of their physical properties.
3. Chemotherapy—cytotoxic agents acting by virtue of their chemical properties.
4. Hormonal therapy (additive or ablational)—acting as environmental alterants.

Let us analyze each of these, and see wherein it affords palliation.

A. SURGERY

Surgical procedures in palliation aim to

accomplish three things:

1. *Removal of a fungating, painful, or compressing tumor mass.*

Examples are many: Simple mastectomy, nephrectomy, partial thyroidectomy, extremity amputations, wedge resections of intestinal lesions, partial gastrectomy. These procedures are frequently carried out in the face of demonstrable distant metastases for the purpose of removing a disabling, painful, or disfiguring tumor or to prevent complications from its continued growth. A prime example of surgery for palliation is simple mastectomy in a case with known distant metastasis. It has been shown [8] that in these cases a relatively simple procedure produces subjective and objective improvement with added survival.

2. *Relief of complications*:

Here we include colostomy for intestinal obstruction, gastroenterostomy, gastrostomy or ileostomy to overcome high alimentary tract obstruction, tracheostomy to afford an open airway, craniotomy and laminectomy to relieve pressure, and the treatment of pathologic fractures.

3. *Relief of pain*:

These neurological procedures are effective in making the patient more comfortable and in minimizing the necessity of narcotics and their consequent bad effects. These procedures include rhizotomy, peripheral neurectomy, chordotomy, lobotomy.

4. *Hormonal ablational*:

These are oophorectomy, orchiectomy, adrenalectomy, hypophysectomy.

It is impossible to discuss all of our attainments in each of these fields. The hormonal ablational effects will be discussed in conjunction with hormone therapy. But for purposes of illustration, we will discuss results of surgical palliation in certain specific lesions.

The statistical approach to the evaluation of surgery in palliation is fraught with many difficulties. There are no controls; there is no standardization of staging permitting a fair comparison of different procedures in similar malignancies, and surgical abilities and techniques differ markedly. Despite these obvious facts which make any statistical evaluation difficult, one may be justified in attempting such a study in order to gain an impression, at least, of the relative value of certain palliative operations. We are reminded of E. D. Churchill's comment: "Opinions and impressions are plentiful and cheap . . . facts are rare and precious."

1. Partial gastrectomy in incurable pyloric cancer.

Our experience at the Conferedate Memorial Medical Center in the palliation of incurable gastric carcinoma is somewhat similar to that reported by Pack and McNeer.[28] In a series of 100 cases of gastric carcinoma collected [36] in the years 1940-1954, it was found that following palliative partial gastric resection there was an average postoperative duration of life of seventeen months as compared to the procedure of gastroenterostomy and gastrostomy in which all patients were dead within six months. Obviously, palliative resection was carried out in generally more favorable cases than the other two procedures. Nevertheless, it is the general consensus that the best palliation for gastric carcinoma is removal of the primary lesion if at all possible. The operative mortality in our series of palliative resections was 5.4 per cent. This mortality should not mitigate against advocacy of palliative gastric resection.

Of a total of 70 gastric resections done in the Confederate Memorial Medical Center over the stated period of time 52 per cent were palliative. In a group of 58 colon resections done between 1940-50 [5] 34 per cent were palliative.

2. Palliative surgery in carcinoma of the head of the pancreas.

Cancer of the pancreas is about as incurable a disease as any to which man is heir, either due to difficulties in early detection or the problems in surgical extirpation. Yet the patient is not to be abandoned therapeutically, for studies indicate that there is a definite prolongation of life by palliative procedures and in those cases with obstructive jaundice, the patient is more comfortable after his jaundice is relieved by a by-pass procedure.

In a collected report by Cliffton,[9] he found that the average survival after diagnosis of carcinoma of the pancreas, without treatment, ranged from three to ten weeks. Those cases having palliative radiation, survived an average of eight months. Those having palliative by-pass procedure survived 5.4 to 13.9 months, while those undergoing excisional surgery survived 10.5 to 25.8 months. Obviously, in the first group were the more advanced cases, in the last, those in which a cure was sought. But the cure rate is so infinitesimal that for all practical purposes, palliation was all that could be expected and apparently to some degree this aim was realized. Obviously though, the more extensive procedure has a much higher mortality which might encourage one to approach the problem of palliation with a less formidable surgical procedure.

There are certain other surgical procedures which have been advanced for supposedly giving a higher cure rate in certain malignancies. But because of the distressingly low improvement in cure rate in most of these, and the questionable palliation afforded, they are debatable procedures. Such are:

1. Multiple organ excision and pelvic eventration where the lesion has extended to adjacent organs.
2. Total gastrectomy.
3. Quarterectomy for extremity malignancies.

The surgeon carrying out these procedures is often like the man who marries for a second time: It is triumph of hope over experience.

B. RADIATION THERAPY

Radiation therapy has been available for many years, but in recent years we have seen the development of many improvements in methods and newer sources. Methods thus far are of four types:

1. *External ionizing radiation*:
   a. X-rays from 50,000 to 3,000,000 volts
   b. Radium
   c. Radio-isotopes, e.g., Co^{60}, Ce^{137}
   d. Electron, proton, neutron beams
2. *Intracavitary radiation*
   a. Radium or radon
   b. Radio-isotopes, e.g., Co^{60}, P^{32}, Na^{24}, Au^{198}
3. *Interstitial radiation*
   a. Radium, radon
   b. Co^{60}
   c. Au^{198}
4. *Internal or parenteral irradiation*
   a. P^{32}, I^{131}, Na^{24}, Au^{198}

In general, it may be said that the newer methods of irradiation have not yet been demonstrated to increase the cure rate, but they have been a valuable addition to palliative therapy. To give certain examples: The use of the cross-fire technique and cobalt beam therapy has permitted regressive doses of x-ray to internal malignancies, with less systemic and local discomfort to the patient. While not apparently increasing the cure rate, it has the palliative value of making the patient less uncomfortable from radiation complications.

Au^{198} has had beneficial effect, albeit, temporary, in ascites, pleural effusions.

I^{131}, with or without preliminary thyroidectomy, has been of value in treatment of some metastatic lesions of thyroid cancer, and occasionally of the primary lesion, and perhaps in prolonging life. However, it should be remembered that less than 50 per cent of thyroid cancers will concentrate a significant amount of I^{131}. Fewer than 25 per cent of papillary adenocarcinomas (the most frequent type) will retain I^{131}. More malignant forms concentrate very poorly. Questionable palliation in breast cancer with P^{32} (and testosterone) has been reported. X-radiation in pathological fractures is of proven benefit.

There can be no doubt that radiation therapy can cure certain accessible lesions as of the skin, breast, and cervix; that the purposes of palliation, i.e., prolongation of life and relief of disabling symptoms can be afforded in certain selected and suitable lesions, and that with improvements in technique and newer methods, even further palliative and perhaps curative results may be forthcoming. Radiation, too, has its dangers: pulmonary fibrosis, depression of

bone marrow, necrosis and skin irritation, ureteral blockage.

C. CHEMOTHERAPY

Perhaps the most promising of all the newer methods of cancer therapy have been in the field of chemotherapy. Certainly it is the field in which the ultimate "break through" in the control of cancer is expected. No chemotherapeutic agent has yet been demonstrated to have cured any patient of cancer. However, such substances may relieve symptoms, reduce periods of hospitalization, prolong life, and increase probability of cure by other means. The effect of these various chemicals on cancer has been partly discovered by accident, partly as the result of definite and specific research. They work in various ways, but none, as yet, has been found that meets the ultimate: interferences or destruction of cancer cell growth without adversely affecting normal cells. There are five types of drugs now in general use. So far, none is curative; their greatest benefit has been in palliation of certain lymphomas and leukemias.

However, the use of chemotherapeutic agents in the prophylaxis against metastasis at the time of surgery opens an intriguing field. Cole and associates [14] have shown experimentally that injection of tumor cells and certain chemicals at the same time reduces markedly the development of metastatic lesions. They are trying it clinically in primary operable lesions on the breast, colon, rectum, and stomach, using the drugs (HN_2 and TESPA), locally and intravenously immediately following and for three days postoperatively, in the hopes of destroying cancer cells in transit.

1. *Nitrogen mustard-type compounds*:
   a. *Nitrogen mustard* (NN_2)

It probably acts at the point of metabolism of nucleic acid, causing mitotic inhibition. This drug has given definite remissions and palliation in the following conditions:

(1) *Hodgkin's disease*: 80 per cent show remission at some time or other. It does not increase longevity, but will cause decrease in fever, relief of pain, reduction in size of tumors, and will apparently convert an acute fulminating Hodgkin's to the chronic phase.

(2) *Lymphosarcomas*: There is a similar response as in Hodgkin's; life expectancy is not increased but makes life more bearable. Some types e.g., reticulum-cell respond poorly.

(3) *Leukemia*: HN_2 is of *no* value in acute leukemia; it is of moderate value in chronic myeloid and lymphoid leukemia: it is unpredictable in its effects.

(4) *Bronchogenic carcinoma*: There has been sufficient benefit here to make it of definite value in palliation. The more anaplastic the carcinoma, the better the results. *75 per cent* of incurable bronchogenic carcinomas show remission of symptoms lasting from two weeks to two months with alleviation of cough, pain, dyspnea, hemoptysis.

(5) *Metastatic carcinoma*: HN_2 sometimes gives temporary relief in breast, ovary, uterine, gastric malignancies, seminoma, chorio-epithelioma. Its effect is sporadic and unpredictable.

b. *Triethylene melamine (TEM)*

It is a nitrogen mustard-like compound and closely resembles HN_2 in its therapeutic and toxic effects. Its main advantage is that it is effective when given by mouth and there is less nausea and vomiting. It is as effective in some cases as HN_2 and because of less toxicity can sometimes permit continuation of therapy and thus prolong remissions.

c. *Triethylene phosphoramide (TEPA)* and d. *Triethylene thiophosphoramide (TESPA)*

Both have similar actions but TESPA is less toxic and better tolerated. Therefore, TESPA is used more frequently. It may be used intravenously, intramuscularly, interstitially, intracavitarily. Variable palliation is given in breast and ovarian carcinoma and in chronic lymphatic and myeloid leukemia.

2. *Urethane*:

Thirty-five per cent of *multiple myeloma* cases are favorably affected, but it is not known that it increases longevity. It causes remission in *chronic leukemias* and is more effective in chronic myeloid leukemia, but is no better than x-ray or P^{32}.

3. *Folic acid antagonists* (aminopterin and A-methopterin)

Its use is predicated on the fact that folic acid is essential for cell metabolism. It is best in *acute leukemia*: One-third show good response. Remissions are temporary but have extended survival time of those who have responded up to twelve months.

4. *Compound 6—mercaptopurine*:

It is a purine antagonist and is of value in *acute myeloid leukemia*. Remissions are temporary, lasting two to eight months.

5. *Myleran (GT-41)*

This is a sulfonic acid ester and can cause marked depression of granulopoiesis. It does not appreciably affect the lymphatic system. Its principal use is in *chronic myelogenous leukemia* Remissions become increasingly shorter, but it can cause definite remissions up to a year's duration.

D. HORMONAL THERAPY (ADDITIVE AND ABLATIONAL)

One of the most fruitful fields of cancer palliation has been the use of hormonal therapy, either as an additive procedure, or by ablation of certain of the endocrine glands. Hormones act as regulators of growth processes and modifiers of cellular enzymatic actions. The fact that we can, even temporarily, favorably affect the course of a malignancy is stimulating and promising of yet greater knowledge and accomplishments to come.

To date, palliation by hormones, specifically the sex steroids, estrogens and androgens, has, not surprisingly, been most effective in sex-linked organs and in particular the prostate and breast. Since principal accomplishments have been in these two organs, we will confine our remarks to them.

1. *Prostate*:

Most cases of carcinoma of the prostate when first seen usually have progressed to the stage of being surgically incurable, i.e., they have extended beyond the prostatic capsule. The value of estrogen therapy and/or orchiectomy in producing prolonged remissions in prostatic cancer has been one of the most valuable of palliative procedures. Some have even suggested that by use of these palliative measures, a previously surgically incurable lesion can be converted into a surgically curable one.[10, 35] Prostatic cancer has been controlled by palliative measures for five years and more. In the presence of bony metastasis, it takes five to six months to see satisfactory regression.

In a study by Pool and Thompson [31] they found in a collected series of 1560 cases of prostatic carcinoma, all treated with TUR but in which estrogens were administered in a group of 649, that there was a 12.1 per cent greater five-year survival rate in the estrogen treated group. Since after removal of the sources of androgen (orchiectomy) and anti-androgen therapy (estrogens), the adrenals are capable of compensating and producing androgens in significant amounts, failure of further palliation after orchiectomy and estrogen therapy can be combated for three to nine months further by bilateral adrenalectomy. Hypophysectomy causes some palliation but its value remains to be determined.

2. *Breast*:

Confining our remarks to the female breast, effective and real palliation in surgically incurable breast cancer has been afforded through:

(1) Local surgery
(2) Irradiation—x-ray—radium—P^{32}, Au^{198}
(3) Chemotherapy
(4) Hormonal therapy
    a. Ablational
    b. Additive

We have already touched upon the use of surgery, irradiation, and chemotherapy in palliation of breast cancer. In recent years, the hormone approach has been intensely studied by a group-study sponsored by the A.M.A. Committee on Research. That study was recently concluded after work of about 30 groups, of which we were for-

tunate in being one. Let us then summarize the current views concerning hormone therapy in palliation of female breast cancer.

Ablational hormonal therapy: The earliest linking of hormones and cancer therapeutically was in cancer of the breast. Beatson,[4] in 1896, reported on the beneficial effects of oophorectomy in the palliation of extended breast cancer. In 1905, Lett [22] collected 99 cases, 46 per cent showing a favorable response. Treves and associates [37] at Memorial Hospital have shown that 44 per cent of premenopausal women have subjective and objective improvement, lasting from eleven months to two to three years. Only 12 per cent favorable response was noted in postmenopausal women. That surgical castration is more effective than radiation castration is indicated by 18 per cent further response in the cases oophorectomized following radiation of the ovaries. There can be no question but that in premenopausal women, who have recurrence or generalized extension of their breast cancer, the most effective method of palliation is initially bilateral oophorectomy.

There has been increasing evidence suggesting that oophorectomy should be part of the primary treatment in premenopausal women with operable breast cancer, concomitant or immediately following radical mastectomy. Horsley [21] has reported increased five-year survival in 9 to 12 per cent of such cases, and Treves, from Memorial, has similarly found the combined procedures to increase five-year survival (10 to 20 per cent). Here is, again, partially a question of philosophy: Treat cancer of the breast piecemeal, or hit it with all we have initially. We have, for several years, routinely elected to do bilateral oophorectomy at the time of the initial treatment, but it is too early to evaluate our results.

It is apparent that there are two types of breast cancer, not predictable by histologic or other methods at present: one is apparently estrogen dependent, the other not. The only way, thus far, to distinguish the two is by the effect of oophorectomy or estrogens. Those that are estrogen dependent are the ones responding favorably to oophorectomy. After removal of the ovaries, the adrenals compensate and secrete estrogens. In those who have responded to oophorectomy and then begin to regress, further regression in 42 per cent for a nine months' period can be expected by bilateral adrenalectomy. Similar response can be expected from hypophysectomy (surgical or irradiation). Some favor hypophysectomy to adrenalectomy because of the easier postoperative control. In postmenopausal women who have had recurrence of their cancer, around 60 per cent objective improvement lasting from nine to ten months has been observed following combined bilateral oophorectomy and adrenalectomy. "Medical" adrenalectomy, i.e. suppression of adrenals with cortisone, has not been as effective as adrenalectomy. Objective remission has occurred in only 15 per cent of cases.

Additive hormonal therapy: Both estrogens and androgens have been valuable adjuncts in the palliation of breast cancer. This mode of action is unknown, but may be due, as suggested by Nathanson, to hormonal or suppressive hypophysectomy.

Estrogen therapy: Estrogen therapy is the more effective of the two types of steroid therapy (i.e. estrogens and androgens) but since it can often accelerate the disease in the presence of functioning ovaries, it is reserved for women who are six years clinically postmenopausal or six months or more surgically postmenopausal. It is equally effective as androgens in bony metastasis and twice as effective in soft tissue metastasis. It takes two to three months to effect palliation and effective palliation can be produced in 30 per cent of cases for periods of six months up to several years.

Androgen therapy: This is not as effective as estrogen therapy, but can be used in premenopausal women safely. It affords something less than 30 per cent palliation and the period of palliation seems somewhat shorter.

SUMMARY AND CONCLUSIONS

1. The over-all cure rate in all types of

cancer is 25 per cent; 75 per cent will require palliative treatment at some time in the course of their disease.

2. Effective and real palliation can be had in about 75 per cent of those requiring it.

3. Advances in palliation are an important aspect in cancer management; the cure rate is not the only index of progress in this field.

4. The purposes of palliation, in effect, approximate our primary function as physicians: We do not expect to banish death from the earth, but only to prolong life and to make that life more comfortable.

5. The purposes of palliation are accomplished by (a) temporary regression or cessation of tumor growth, (b) relief of disabling or lethal complications, (c) giving symptomatic relief.

6. In addition to general supportive measures, effective palliation can be afforded in selected cases by (a) surgery, (b) radiation, (c) chemotherapy, (d) hormonal therapy.

7. Surgery and radiation are curative as well as palliative modalities. Chemotherapy and hormonal therapy are palliative in selected cases, but so far neither has proven of curative value.

8. Surgery and radiation therapy are effective in a wide range of malignancies. Chemotherapy is principally of value in the lymphomas and leukemias. Hormonal therapy is principally of value in prostate and breast malignancies.

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THE PRESENT STATUS OF THE TREATMENT OF DISEASED BLOOD VESSELS BY REPLACEMENT *

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Within the past decade a variety of vascular substitutes have been developed for replacing segments of the aorta and peripheral arteries. Homografts have been utilized extensively with considerable success and both early and late results indicate that homologous arteries, whether fresh, preserved by refrigeration of by freeze-drying, are satisfactory arterial replacements.[3] Recent observations of atherosclerotic changes in aortic homografts one or two years after transplantation suggest that these grafts may undergo degenerative changes with time,[1] but additional studies are needed to support these observations.

Because of the difficulty in procuring arterial homografts experimental and clinical trials of heterologous grafts have been recorded. The rapid degeneration of these transplants, however, indicates that they are entirely unsatisfactory for arterial replacement.

In view of the difficulties associated with homografts, vascular prostheses made of synthetic materials have been extensively studied. To date, nylon, dacron, orlon, and teflon appear to be most satisfactory for arterial substitutes. Porous and nonporous materials appear to function equally well in the aorta. For peripheral vessels, however, the crimped nylon tube developed by Edwards and Tapp[6] has proven most effective. Recent modifications of this type of crimped nylon tubing have been made available which incorporate bifurcations. This has extended the range of their usefulness enormously. With increasing experience in the use of these nylon prostheses they are found to be satisfactory substitutes in a variety of aneurysmal and occlusive diseases at all levels of the arterial system. At the present time the only sites where homografts have a distinct advantage is at the levels of elaborate branching, particularly the aortic arch and the upper abdominal aorta. Crimped nylon tubes are now employed by preference for peripheral arteries, the lower abdominal aorta, the descending thoracic aorta, and the ascending aorta. Morphologic studies up to two years after transplantation fail to reveal degenerative changes in synthetic arterial substitutes.

The indications for use of arterial replacement have rapidly increased to the point that practically every segment of the aorta and major peripheral arteries has been successfully replaced. This report is concerned with a brief review of the indications and general principles underlying the use of arterial substitutes in the treatment of acquired diseases of the arterial system.

Aneurysms and atherosclerotic occlusive disease are the two most common acquired diseases of the aorta. Unless treated, these conditions are associated with progressively severe symptoms, and in a significant number of cases result in fatal complications. In the case of aneurysms, death occurs usually from rupture and hemorrhage, the average duration of life after diagnosis being about one to two years. Thrombotic occlusion of the aorta and peripheral arteries runs a more protracted course but the final complications of arterial insufficiency may lead to death.

Dissecting aneurysm of the aorta is a relatively common condition which is rapidly fatal in about 90 per cent of cases unless treatment is instituted early.

Traumatic rupture of the thoracic aorta is occurring with increasing frequency as a result of the increase in automobile accidents. This condition may not be immediately fatal and recent experience indicates that sufficient time may elapse between injury and fatal hemorrhage to

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permit operative treatment.

ANEURYSMS OF THE AORTA

Aortic aneurysms may be sacciform or fusiform in shape and result from atherosclerosis, syphilis, or trauma. As a rule sacciform aneurysms are due to syphilis, the characteristic pathologic feature being a destruction of the elastic tissue with weakening of the wall. If the destructive process is localized the dilatation may involve only a portion of the circumference of the aorta. In many instances the resulting saccular aneurysm has a small neck. This type of aneurysm is most commonly found in the thoracic aorta, in the ascending portion an the aortic arch (Figure 1). Symptoms may not appear until late and usually are a result of compression of adjacent structures often with erosion into vertebral bodies, trachea, or esophagus.

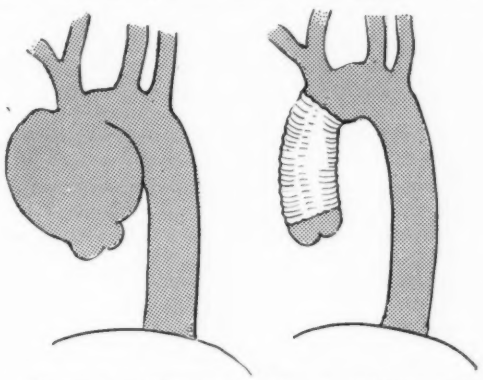

Figure 1.—Diagram showing a fusiform aneurysm of the ascending aorta which was resected using extracorporeal circulation and replaced with a crimped nylon tube.

The characteristic pathologic feature of a narrow neck at the site of origin of these lesions makes possible the removal of the aneurysm by occlusion of the neck and excision followed by repair of the aortic wall. Thus this can be accomplished in most instances without encroaching upon the aortic lumen.

Fusiform aneurysms involve the entire circumference of the aorta. Generally, fusiform aneurysms of the thoracic aorta are syphilitic or traumatic in origin while those involving the abdominal aorta are due to atherosclerosis. Another interesting feature of these lesions is that aneurysms of atherosclerotic origin almost invariably involve the portion of the aorta below the renal arteries. As in the case of sacciform aneurysms, these lesions produce symptoms by pressure on adjacent structures with back pain being a common feature. This is often mistaken for conditions involving the intervertebral disks or lesions of the skeletal system.

Regardless of their location, fusiform aneurysms are best treated by complete excision and replacement with an arterial substitute. Since it is necessary to interrupt blood supply to several organs during operation the technic employed depends upon the location of the aneurysm. For instance, in aneurysms of the ascending aorta and aortic arch, it is necessary to maintain blood flow to the central nervous system during the period of aortic occlusion. This can be readily accomplished by use of temporary bypass shunts or by utilizing a mechanical heart-lung bypass. The former procedure is especially useful when the aneurysm does not involve the ascending aorta and there is sufficient aortic wall above the valve to implant a shunt. However, should the aneurysm arise in the ascending aorta, the most practical technic consists of the use of a mechanical heart-lung bypass. In this procedure blood is removed from the superior and inferior venae cavae, pumped through a mechanical oxygenator, and then returned to the common carotid arteries and to one of the femoral arteries. Thus, adequate blood flow to the brain and to structures below the aortic arch is maintained. When aneurysms involve the descending thoracic aorta, ischemic damage to the spinal cord may result from occlusion of this portion of the aorta (Figure 2). Therefore, it is necessary to either reduce the metabolism of the spinal cord by reducing the temperature or use temporary bypass shunts. General body hypothermia has been widely employed and is a relatively safe and effective technic for this purpose. It is now recognized that

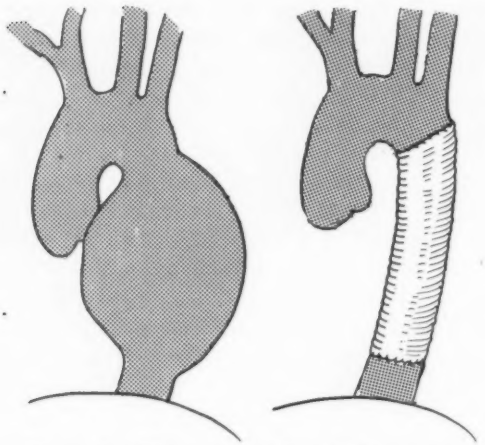

Figure 2.—Diagram showing fusiform aneurysm of the descending thoracic aorta which was resected under hypothermia and replaced with a nylon graft.

the temperature need not be reduced to extremely low levels but only to about 80° to 90° F. Temporary bypass shunts are also quite effective in preventing ischemic damage to the spinal cord and have certain advantages over general body hypothermia. These shunts may extend from the left atrium of the heart through a pump and into a femoral artery, or from the left subclavian artery to the left femoral artery. In either technic, the thoracic aorta may be cross-clamped as long as ninety minutes with assurance that the spinal cord and kidneys are receiving adequate blood flow.

Fusiform aneurysms involving the abdominal aorta below the renal arteries are less of a problem since the aorta in this area may be occluded for as long as two hours without producing ischemia to the distal tissues. Thus, aneurysms in this location may be resected with relative ease and replaced with some type of arterial substitute.

LOWER THORACIC AND UPPER ABDOMINAL ANEURYSMS

One of the most difficult aneurysms to treat effectively is that involving the lower thoracic and upper abdominal portions of the aorta (Figure 3). This is true because the vital visceral branches of the aorta arise from these segments. Therefore, in resecting aneurysms in this area it is necessary to employ temporary bypass shunts in order to minimize the period of interruption of blood flow through the celiac, superior mesenteric, and renal arteries. Increasing experience has indicated, however, that with the use of shunts interruption of blood flow to these

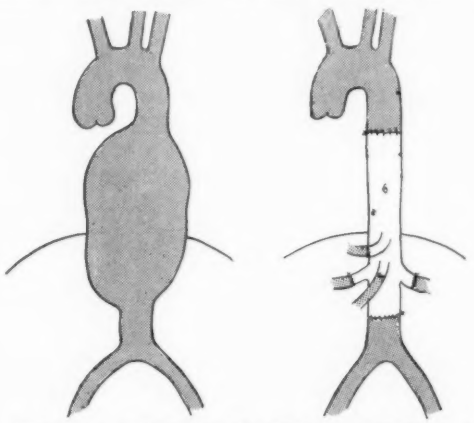

Figure 3.—Diagram showing an aneurysm of the lower thoracic and upper abdominal portions of the aorta with involvement of the celiac, superior mesenteric and renal arteries. The aneurysm was resected using a temporary bypass shunt and the defect bridged with a homograft with restoration of continuity of the involved visceral branches.

organs can be reduced to a safe level with only minimal, temporary interference with function.[5]

RUPTURED ANEURYSMS

Rupture of an abdominal aneurysm represents a catastrophe, yet because of the pathologic anatomy of abdominal aneurysms it is frequently not immediately fatal. Thus, rupture does not usually take place as a sudden event because of the retroperitoneal position of the aneurysm and the characteristic lamination of the thrombus attached to its wall. The clinical manifestations consist of onset of abdominal pain, tenderness over the aneurysm or in the flanks, and a moderate reduction in blood pressure. In some instances, compression of one or both ureters may produce symptoms suggestive of ureteral calculi. In many cases symptoms will abate after the initial episode but

rupture then occurs hours or days later. In the presence of an abdominal aneurysm, the symptoms described above should suggest the diagnosis and treatment should be instituted promptly. Operation consists of removal of the ruptured aneurysm and replacement with a suitable substitute.

The extremely poor natural prognosis of aneurysms of the aorta makes operation imperative unless the patient's condition contraindicates it. In an experience consisting of more than 300 cases, the mortality of patients without significant cardiopulmonary or renal disease is about 10 per cent.

DISSECTING ANEURYSM

The term "dissecting aneurysm" is often a misnomer since the actual diameter of the aorta may not be increased. The diagnosis can usually be established from the clinical manifestations alone which consist of sudden onset of severe excruciating pain beginning in the chest and radiating peripherally into the upper extremities or into the abdomen and lower extremities. In most instances symptoms are suggestive of coronary occlusion but a negative electrocardiogram helps to rule out this condition. The dissecting process usually begins in the thoracic aorta just above the aortic valve or just distal to the left subclavian artery and may extend as far distally as the popliteal arteries.

The etiology of the condition is not clear but in most instances there is degeneration of the medial coat and the patients are usually moderately to severely hypotensive. Only about 10 per cent of patients survive the acute dissecting process and then only because the dissection ruptures back into the true aortic lumen. This has been referred to as a "healed" dissecting aneurysm. The remainder of these cases terminate by rupture through the adventitia and then into adjacent structures such as the pericardial sac, mediastinum, or pleural cavities.

Recently, technics for the treatment of dissecting aneurysms have been developed which are based upon nature's method of healing this lesion.[2] Thus, when there is simple dissection of the aortic wall without aneurysm formation and the process begins in the ascending aorta, treatment consists in creating a window in the dissected portion of the wall a short distance below the origin of the left subclavian artery to provide a re-entry passage to the true aortic lumen. When the dissecting process begins at the left subclavian artery and the aortic wall is dilated, this segment of aorta is resected using either hypothermia or a bypass shunt. The dissected passage distally is then obliterated by sutures and the excised portion of aorta is replaced with an arterial substitute. The operative mortality is about 20 per cent but this compares favorably with the natural mortality of this disease of 90 per cent.

ANEURYSMS OF PERIPHERAL ARTERIES

Although aneurysms most commonly involve the aorta, their occurrence in peripheral arteries is not infrequent. In most instances peripheral aneurysms are a result of atherosclerosis or trauma. The most common sites for the former are the femoral artery in the region of the inguinal ligament and the popliteal artery. Rarely do arteriosclerotic aneurysms occur in the vessels of the upper extremities. Traumatic aneurysms may occur at the site of any penetrating arterial wound. These lesions may be confined solely to the artery or may involve both artery and vein with a communicating arteriovenous fistula. Peripheral aneurysms are best treated by total excision and replacement with a vascular substitute. The results are eminently satisfactory and, in the case of arteriovenous aneurysms, have rendered obsolete the procedure of quadruple ligation and excision without restoration of continuity of the artery.

OCCLUSIVE DISEASE

Atherosclerotic, thrombotic occlusions characteristically involve the abdominal aorta near its bifurcation and the vessels distal to this site. The occlusive process may be partial or complete and in most instances is segmental. As a rule, partial occlusion of the aortic bifurcation is asso-

ciated with involvement of the vessels of the lower extremities as well; whereas, in complete aortic occlusion (Leriche syndrome) the vessels of the lower extremities are less often affected. When the occlusive process involves the abdominal aorta, symptoms of imminent claudication and weakness involve hips, thighs, buttocks, and calves. On the other hand, if the occlusive process involves the external iliac or femoral arteries, symptoms are confined to the calf muscles. In the Leriche syndrome with complete aortic occlusion, hypertension is a common feature and some degree of sexual impotence is almost invariably present. This type of the disease appears in the middle decades; whereas the partial aortic occlusion and peripheral arterial diseases are more common in the later decades.

Diagnosis can be made on the basis of clinical manifestations alone but should be confirmed by visualization of the arterial tree through the use of arteriography. This diagnostic procedure has made it quite clear that, in the majority of instances, atherosclerotic occlusion is segmental in nature. Thus, in patients with absent pulses in the lower extremities, segmental occlusion of the aortic bifurcation and common iliac arteries can be anticipated; whereas, in the presence of femoral arterial pulsations but absent pulses below this site, segmental occlusion is located in the superficial femoral artery. When a radio-opaque medium is injected proximal to the site of occlusion and x-rays are made at appropriate intervals, the site of the occluded segment can be demonstrated and the patent vessel below the site of occlusion becomes filled with dye through collateral branches and is visualized also.

Atherosclerosis is a generalized disease and surgical correction of an occluded blood vessel will have no effect on the course of the disease. However, by restoring normal blood flow to parts distal to the block, it is possible to completely relieve symptoms, thus permitting many patients to return to work. There are two operative procedures available for use in treating this disease. The first consists in excising the involved vessel longitudinally and removing the occluded thrombus and attached atherosclerotic intima. This procedure is known as thrombointimectomy. It is useful particularly for occlusions of limited extent and has the advantage that it permits restoration of blood flow without insertion of foreign material. The second type of surgical procedure consists in excision of the involved artery and replacement with a substitute or bypass of the occluded segment. The former is useful primarily in the treatment of occlusive lesions of the aortic bifurcation; whereas, the bypass procedure is most often employed in the treatment of occlusions of the femoral artery. Here, the involved vessel is undisturbed thus minimizing the likelihood of damage to functioning collaterals. The ends of the arterial substitute are anastomosed to the side of the artery above and below the site of occlusion thus providing, in effect, a large, new collateral (Figure 4). Although arterial homografts and autogenous vein grafts have been used extensively for this purpose, recently it has

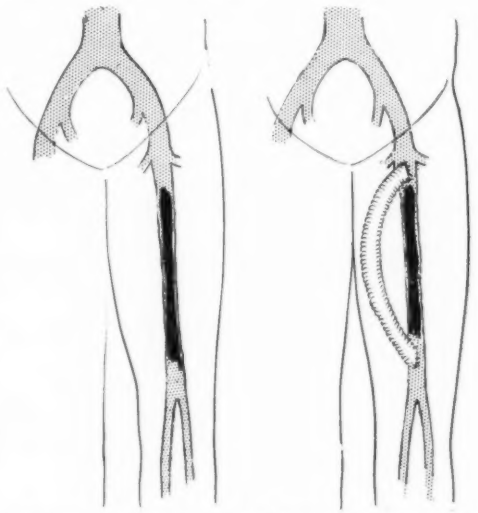

Figure 4.—Diagram showing segmental occluson of the left superficial femoral artery treated by the bypass procedure using a crimped nylon graft.

been demonstrated that the crimped nylon tube is eminently satisfactory for this type of operation.

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VALUE OF METRAZOL THERAPY IN THE AGED *

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INTRODUCTION

Because of lengthening life span and increased tensions associated with modern urban life, the present-day physician is more frequently faced with the problems and treatment of degenerative diseases than was his forefather. Diseases associated with aging involve several organ systems; for example, those emanating from arteriosclerosis and the wear and tear of everyday life. Consequently, the physician faces a constantly increasing number of geriatric patients. Many recent studies have been directed toward combatting the major problems of aging.[1-3] I feel the answer lies in an agent that will retard the aging process and one that can be used satisfactorily for long-range treatment of cases involving senile neuroses such as functional memory defects, mental confusion, mild behavioral disorders, irritability, depression, and antisocial attitudes in the senile.

Metrazol, a stimulant in circulatory and respiratory failure, in collapse, alcoholic poisoning, fatigue, and senile confusion, seemed to me to be the drug of choice. Chesrow, Giacobe, and Wosika [4] reported in 1951 that "the analeptic action of the drug and its ability to increase nerve impulse transmission" acted as a general tonic in 26 of 32 cases of arteriosclerosis, with no significant or unfavorable clinical side effects. For a clinical investigation Smigel, Serhus, and Barmak [5] placed 32 patients on Metrazol therapy; of these 27 tolerated the drug well and showed definite improvement.

PHYSIOLOGY

The physiological action of Metrazol results in increased oxygenation of the blood due to stimulation of the respiratory and vasomotor centers of the medulla.

Cohn and Cohn [6] reported beneficial effects on the entire cerebrospinal axis and postulated that Metrozol exerted its effects primarily on the vasomotor and respiratory centers. The drug promoted glycogen formation in the cerebral cortex and increased cell membrane permeability.

Review of the literature revealed that a general tonic effect is brought by decreasing rather than increasing hypertension. The drug raises blood pressure only in cases of hypotension.

SELECTION OF CASES

Patients to be put on Metrazol therapy were selected in a logical fashion from individuals under my care in nursing and convalescent homes. Metrazol, 6 grains per day, was administered either in tablet or liquid form to 17 patients with cerebral arteriosclerosis, some of whom were psychotic. The individuals varied in age from 64 to 85 years; the median was 75. All exhibited a degree of early, moderate, or advanced arteriosclerosis. Bear in mind that my aim was not to evaluate a drug but rather to improve the mental outlook and nourishment of individual patients. My primary interest is to provide the geriatrician or general practitioner with a drug of proven effectiveness for use in convalescent homes and private practice.

DISCUSSION

Of 17 cases given therapy in convales-

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cent homes, 11 (64.7 per cent) showed definite improvement. Of the 7 cases of early arteriosclerosis, 6 (85.7 per cent) were moderately improved. Of the 6 cases of moderate arteriosclerosis, 4 (66.67 per cent) were definitely improved. The 4 cases of late arteriosclerosis remained unchanged or continued to regress.

I should like to describe the 6 unimproved cases.

TABLE 1
CASES GIVEN METRAZOL THERAPY IN CONVALESCENT HOMES

Initials	Age	Syndrome	Improvement	Drug Given
Case 6	84	Arteriosclerosis; depression	None	6 gr. daily
Case 12	77	Arteriosclerosis	None	6 gr. daily
C. M.	84	Arteriosclerosis	None	6 gr. daily
K. R.	85	Arteriosclerosis; depression	None	6 gr. daily
A. M.	82	Arteriosclerosis; senile psychoses	None	6 gr. daily
M. D.	70	Arteriosclerosis; depression	None	6 gr. daily

Case No. 6 was bedridden. She had a marked cerebral arteriosclerosis with cardiac decompensation, depressive psychosis, diabetes mellitus, fracture of the right acetabulum, and subclinical pellagra. When Metrazol therapy was instituted we hoped that her psychoses, nutrition, and cardiac decompensation would improve. However, after four months of therapy she had not improved and continues on a downhill course.

Case No. 12, also admitted to a convalescent home with diagnosis of psychosis and marked cerebral arteriosclerosis, had a history of prostatectomy one year previous to admission in 1955. He had been treated at a psychiatric institution because of a depressive psychosis. He seemed to have an improvement in appetite, gain in weight, and was better oriented; there was improvement in sensorium. However, there has been a decline in appetite, he is emaciated, has psychosis, and is progressively worse in sensorium. In spite of Metrazol therapy for five and a half months he went into a semicomatose state because of the marked arterial degeneration.

Case No. 13, C. M., age 84 years, was a female patient admitted to the sanitorium with marked cerebral arteriosclerosis and cerebral psychosis. The degree of sclerosis was so marked that she was confused, completely disoriented, unable to answer questions, and was a stuttering and blabbering imbecile. Her appearance was untidy. She soiled herself but was not bedridden. The radial arteries exhibited a degree of "pipe stem" sclerosis. She was put on two teaspoonfuls of liquid Metrazol twice daily, but after four months therapy showed no improvement in physical and mental status. There was no further degree of mental regression but I feel that she is a therapeutic failure.

Case No. 15, K. R., age 85 years, was admitted to a private nursing home with marked cerebral arteriosclerosis and manic depressive psychosis. Her blood pressure was low, 90 to 100 systolic and 50 to 60 diastolic. Nutritional edema of both extremities was present, and the patient was confused, disoriented, abusive and irritable, but cheerful and almost euphoric at times. Personal hygiene was excellent. After a trial of Rauwolfia type drugs and Metrazol which she took from 1935 to 1955 intermittently, because she thought we were trying to poison her, she was placed on liquid Metrazol in her coffee in February of 1956 and took this medication for four months. She showed no appreciable improvement in sensorium or in psychosis, was still abusive, striking nurses at times. Although her nutrition improved, this was accomplished by diet before 1955, so that we cannot say this is a therapeutic success but a dietary one.

A. M., an 82 year male, was admitted to a private sanitorium after hospitalization in a local institution. He had a history of poor appetite, senile psychosis, and moderate cerebral arteriosclerosis. He was apathetic, hostile, disoriented, withdrawn and had been treated with tranquilizing drugs. After one and a half years of this medication, in March of 1956, he was placed on liquid Metrazol, 2 teaspoonfuls twice daily. Although the appetite improved due to four months of Metrazol therapy, there was no clinical improvement and the patient was still mentally disturbed, apathetic, hostile, and withdrawn. Even before Metrazol was administered, however, the patient's physical status was satisfactory, his blood pressure and cardiac condition being good.

M. D., a 70 year female, was admitted to a private nursing home on two occasions, once in 1952 from a mental institution because she showed no positive response to electroshock therapy. She exhibited presenile early arteriosclerosis with depressive psychosis. She was put on Metrazol, 2 tablets twice daily, on two occasions for periods of thirty days. There was no benefit on either occasion. The patient continued to be depressed, withdrawn, and moody.

Of the 11 improved cases, 7 patients exhibited early arteriosclerosis and 4 suffered with moderate arteriosclerosis. All but one of the early arteriosclerotic in-

TABLE 2
CASES GIVEN METRAZOL THERAPY IN CONVALESCENT HOMES

Initials	Age	Syndrome	Improvement	Drug Given
B. L.	68	Arteriosclerosis; epilepsy	Improved	6 gr. daily
F. O.	73	Arteriosclerosis	Improved	6 gr. daily
M. T.	78	Arteriosclerosis	Improved	6 gr. daily
J. L.	73	Arteriosclerosis	Improved	6 gr. daily
T. S.	75	Arteriosclerosis	Improved	6 gr. daily
V. C.	64	Arteriosclerosis	Improved	6 gr. daily
F. B.	82	Arteriosclerosis	Improved	6 gr. daily
M. P.	72	Arteriosclerosis	Improved	6 gr. daily
C. F.	65	Arteriosclerosis	Improved	6 gr. daily
M. O.	80	Arteriosclerosis	Improved	6 gr. daily
A. B.	75	Arteriosclerosis	Improved	6 gr. daily

dividuals had a variety of associated diseases, including heart diseases, arrested tuberculosis, Meniere's syndrome, epilepsy, late manifest lues, and psychoses. Before treatment with Metrazol this group was characterized by antisocial behavior; they were uncooperative, abusive, and depressed. In addition, there were marked emaciation and slight appetite, poor personal hygiene, hallucinations and dizziness, disorientation, and impairment of sensorium.

Metrazol therapy was given from two to sixteen months; the average term of medication was 7.7 months. After treatment the conditions mentioned above were absent or markedly improved. The greatest change occurred in their nutritional habits and mental attitudes.

I would like to describe two representative histories from this series to illustrate the most and least severe cases.

B. L., the most severe case, a 68 year old male, suffered with mitral stenosis, psychoses, grand mal epilepsy, and emphysema in addition to early arteriosclerosis that had been present for seven years. He had been hospitalized for twenty-five months and received Metrazol for fifteen months. Before treatment he showed marked emaciation, disorientation, hallucinations, frequent grand mal seizures, and was uncooperative. After treatment he did not exhibit emaciation, was well nourished, had few hallucinations and fewer grand mal seizures, and was better oriented and more cooperative.

F. O., a 73 year old female, had early arteriosclerosis only and had been ill for two years, during which time she was hospitalized. Symptoms before treatment comprised confusion, palpitation, myocardial pain, loss of energy, poor appetite, and irritability. After two months on Metrazol, her appearance was better, appetite and nutrition were improved, and the patient was more energetic and less irritable.

Of the 4 individuals who had moderate arteriosclerosis, this condition was the primary cause of their hospitalization. Two of the 4 also had coronary disease, psychoses, and nutritional illness. Before treatment obvious symptoms in this group were poor personal hygiene, impaired sensorium, inadequate memory, poor nutrition, and psychoses. Metrazol was administered to this group from four to sixteen months, on an average of eleven months. After treatment with this drug, general improvement of all symptoms was noted.

SUMMARY AND CONCLUSIONS

This report is based on a study of 17 cases treated in nursing and convalescent homes. They presented lesions of arteriosclerosis concomitant with psychological and nutritional disorders. Metrazol therapy was administered in doses of 6 grains daily. Eleven of the 17 (64.7 per cent) showed definite clinical improvement. The 6 unimproved patients were markedly arteriosclerotic and had other and many serious complications. Of the 11 improved cases the average length of therapy was nine months, which fact would point up the need of long-term therapy before marked change can be anticipated. A small maintenance dose proved sufficient to produce an excellent therapeutic response. In my group of patients the most dramatic improvement was in mental outlook. Extensive destruction to brain tissue as a result of the arteriosclerosis was probably responsible for the lack of improvement in the 6 cases cited. One great advantage of using Metrazol was that no side effects were evident after long-term therapy. Certainly use of this drug seems

a step forward in retarding the aging processes.

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ABO INCOMPATIBILITY

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JAMES W. COTTER, M. D. *
SHREVEPORT

Hemolytic disease of the newborn resulting from incompatibility of the major blood groups is being recognized with increasing frequency. It was demonstrated by Halbrecht [1, 2] and strengthened by Hsia and Gellis [3] that hemolytic disease due to ABO incompatibility is more common than that due to Rh incompatibility.

Although the pathogenesis of hemolytic disease of the newborn due to ABO incompatibility is not completely understood, the current concept is that the disease apparently results from a paternally inherited blood group factor. These factors referred to as 'A' or 'B' are not solely [4, 5] limited to the red cells but are secreted by all the tissues of the affected fetus. After crossing the placental barrier, these highly potent antigenic factors stimulate the production of antibodies by the mother. Witebsky [6] has demonstrated that these antibodies are notably different from the normally circulating 'Anti-A' and 'Anti-B'. They are serum or albumin-active antibodies which are resistant to neutralization by the 'A' or 'B' blood group specific substance. These abnormal antibodies in turn cross the placental barrier and produce a hemolytic process in the infant.

Hemolytic disease of the newborn due to 'A' or 'B' incompatibility is usually not anticipated and is diagnosed after the appearance of clinical jaundice within the first twenty-four to thirty-six hours. In our experience, the disease is less severe than that caused by Rh incompatibility and rarely is a cause of hydrops fetalis.[7] There is no demonstrable relationship between the occurrence of the disease and previous pregnancies or blood transfusions. First-born infants are affected as often as infants born to mothers with normal antecedent pregnancies. There is usually mild or no anemia present with the onset of jaundice. Spherocytosis is always present and associated with an increased fragility of the red blood cells. The direct Coomb's test is usually negative but may on occasion be weakly positive. The ABO Coomb's test is positive in approximately 50 to 60 per cent of cases. In our recognized cases, the mothers' blood type has been 'O' and the in-

TABLE 1.
GENERAL FEATURES OF ABO AND Rh HEMOLYTIC DISEASE OF THE NEWBORN

	ABO	Rh
Prenatal diagnosis	Not anticipated	Anticipated
Severity	Mild	Severe
Onset of icterus	24-36 hours	Birth—24 hours
Hepatosplenomegaly	Mild or absent	Usual
Anemia	Mild or absent	Severe
Hydrops fetalis	Rare	Common
Number of pregnancy	Not related	Increase in severity with succeeding preg.
Spherocytosis	Marked	Unusual
Osmotic fragility	Marked	Seldom found
Coomb's test	Negative, may be weakly positive	Positive
ABO Coomb's test	Positive in 50-60%	——
Major blood type	Related	Unrelated

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fants' blood type has been either 'A' or 'B'. Hepatosplenomegaly is mild or absent.

REPORT OF CASES

Clinical material for this report was taken from a review of cases from Confederate Memorial Medical Center Newborn Nursery and includes material beginning January 1, 1955, and ending March 31, 1957.

TABLE 2.
LIVE-BORN ADMISSIONS TO NEWBORN NURSERY JANUARY 1, 1955, THROUGH MARCH 31, 1957

Total	Nonwhite	White	Ratio of Nonwhite to White
9894	8933	961	9.1

TABLE 3.
PER CENT OF HEMOLYTIC DISEASE IN LIVE-BIRTHS JANUARY 1, 1955 THROUGH MARCH 31, 1957

Total live-births	Occurrence of hemolytic disease	Per cent of total live-births	Rate
9894	38	0.4%	1/260

TABLE 4.
PER CENT ABO AND PER CENT Rh IN TOTAL CASES

Total hemolytic disease	No. ABO	No. Rh	ABO %	Rh %
38	31	7	81.6	18.4

During this period there was a total of 9894 admissions to the nursery. Of these, 8933 were nonwhite and 961 were white. This constitutes a ratio of nonwhite to white of 9 to 1. A diagnosis of hemolytic disease of the newborn was established in 38 of the total admissions indicating an incidence of 0.4 per cent or 1 in 260 live births. ABO incompatibility accounted for 31 or 81.6 per cent of the total. Rh incompatability accounted for the remaining 18.4 per cent. There were 4 or 0.41 per cent ABO incompatibilities in white infants and 27 or 0.30 per cent in nonwhite infants. In 18 cases the mothers' blood type was 'O' and the infants' blood type was 'B'. In 13 cases the mothers' blood type was 'O' and the infants' blood type was 'A'. It was of interest to note that the 4 white infants had blood type 'A'. Of the 31 ABO incompatibilities 19 or 61.3 per cent were treated with exchange transfusion, 3 of which required a second exchange. All 7 of the Rh incompatibilities were treated with exchange transfusion, one of which required a second exchange. One of the Rh incompatibilities occurred in a nonwhite infant.

TABLE 5.
OCCURRENCE BY RACE OF HEMOLYTIC DISEASE

	Rh	ABO	Per cent ABO Occurrence
White	6	4	0.41
Nonwhite	1	27	0.30
Total	7	31	

TABLE 6.
BLOOD GROUPS INVOLVED BY RACE

	Mother 'O' Infant 'B'	Mother 'O' Infant 'A'	Total
White	0	4	4
Nonwhite	18	9	27
Total	18	13	31

ABO incompatibility was recognized in 0.21 per cent of the total births in 1955, in 0.35 per cent in 1956, and in 0.54 per cent thus far in 1957. This suggests an increased awareness and recognition of this entity.

TABLE 7
PER CENT OF ABO INCOMPATIBILITY BY YEAR

	1955	1956	(three months) 1957
Live-born Admissions	4265	4511	1118
ABO Incompatibility	9	16	6
Per cent of total	0.21	0.35	0.54

In a series of 75 cases of ABO incompatibility reported by Leiken and Rheingold,[9] 51 or 68 per cent required exchange transfusion. Of these 51, 34 cases required one exchange; 13 cases required two exchanges: 3 cases required three exchanges; and 1 required four exchanges.

MANAGEMENT

The management of the hemolytic disease of the newborn due to ABO incompatibility depends on several important factors which must be carefully evaluated before definitive measures can be undertaken. They are as follows: (1) the serum indirect bilirubin, (2) proper ABO set-up, (3) hemoglobin, (4) ABO Coomb's test.

Exchange transfusion in these infants hinges on the level of the serum indirect

bilirubin; the critical level being set at 20 miligrams per cent, since the incidence of kernicterus is higher with levels above this figure. There is no adequate explanation as to why kernicterus develops but it is generally felt that the height of the indirect bilirubin, the duration of the level and possibly the rapidity with which it rises are definitely related to the development of such a state. Because of this it is the serum indirect bilirubin which is the most important factor and the one which solely determines whether or not exchange transfusion is to be done.

Anemia may or may not be present in this disease and as a rule, if present, is to a mild degree. Therefore, the infant's hemoglobin in this entity usually plays only a minor role. It must be remembered, however, that a rapid decline in the hemoglobin is indication enough for exchange transfusion in the presence of hemolytic disease.

The ABO Coomb's test is only an adjunct in the diagnosis of hemolytic disease due to this incompatibility and has no part in the definitive treatment. A negative test does not exclude the diagnosis.

In some cases multiple exchange transfusions have been necessary to control the level of the serum indirect bilirubin. This has been true of 3 cases in our ABO series and of 1 of the Rh incompatibilities. It has been our experience that an exchange transfusion in a full term infant utilizes 70 milliliters of blood per pound or approximately 500 milliliters. This amount can be expected to reduce the bilirubin by 60 to 70 per cent. It was recently pointed out that the serum indirect bilirubin after exchange transfusion reaches an equilibrium in four to six hours at two-thirds to three-quarters of the indirect bilirubin prior to exchange.[10] For example, an indirect bilirubin of 20 milligrams per cent prior to exchange is reduced to approximately 8 milligrams per cent. Four to six hours later a level of 14 to 15 milligrams per cent can be expected. After equilibrium has been reached, a continued rise in the indirect bilirubin of 0.5 milligrams per cent per hour is indication for repeat exchange. This was reported by Vaughn and Brilian[5] during the April, 1957 session of the American Academy of Pediatrics.

Exchange transfusions in these infants was done in the usual manner utilizing the umbilical vein. A number 5 polyethylene nasogastric feeding tube has been found to be extremely satisfactory and superior to previously used cannulas. We have found it possible to utilize the umbilical vein as late as five days after birth; however, the average age at which exchange transfusion was performed in the ABO incompatibilities was forty-three hours. On two occasions it was necessary to accomplish the exchange by radial artery and saphenous vein cut-downs. Type 'O' Rh specific blood with added Witebsky substance was used. As previously stated, 70 milliliters per pound is the amount used.

TABLE 8
HEMOLYTIC DISEASE TREATED WITH EXCHANGE TRANSFUSION

	No. ABO	No. ABO Exchanged	No. ABO Not exchanged	No. Rh	No. Rh Exchanged	No. Rh Not exchanged
White	4	1	3	6	6	0
Nonwhite	27	18	9	1	1	0
Total	31	19	12	7	7	0
	61.3 per cent ABO exchanged					
	38.7 per cent not exchanged					

Of the total number of exchange transfusions in ABO incompatibilities in our series, one infant expired during the procedure. This infant was admitted in a moribund condition at the age of five days. Following diagnosis, exchange transfusion was begun two hours after admission. After 70 milliliters of blood had been exchanged, the baby vomited and respiration ceased. Attempts at resuscitation were unsuccessful. No further complications were encountered in the remainder of our series. Careful follow-up in these cases failed to reveal any evidence of kernicterus in these infants.

SUMMARY

Thirty-one cases of hemolytic disease of the newborn due to ABO incompatibility are reported. The disease is being recognized with increasing frequency and is more common than Rh incompatibility. In general the disease is mild; however, 60 to 70 per cent of cases will require at least one exchange transfusion. The disease is not anticipated until the onset of postnatal jaundice. Kernicterus is unusual but high bilirubin levels are as dangerous as with Rh incompatibility.

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VULVAR HEMATOMAS GYNECOLOGICAL AND OBSTETRICAL; REPORT OF FORTY-SIX CASES *

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It is difficult to understand why the American literature and many of the standard American gynecological textbooks are so void on the subject of traumatic vulvar hematomas. When the subject is mentioned the discussion is often short and incomplete. The most significant report is that of Hudock, Dupayne and McGeary [1] who reviewed the literature up to July 1954, and reported 6 cases of their own. A total of 54 cases had been reported, and most of these were by German writers. This is difficult to understand when consideration is given to the fact that these hematomas are not uncommon, are of interesting etiology, rapid in onset, exquisitely painful, very disabling, and that there is little unanimity of opinion regarding their treatment.

This is a report of 21 cases of traumatic vulvar hematomata with an attempt to delineate the best method of treatment as determined by the hospital course and follow-up examinations.

In contradistinction to traumatic hematomas, the hematomata concerned with pregnancy have received a much more thorough coverage in the literature. McElin, Bowers, and Paalman [2] reported 187 cases in a review of the literature up to 1949, and added 73 cases of their own.

This paper will also present 24 hematomas associated with delivery and one occurring spontaneously during the prenatal period.

ANATOMY

The vulva is the external part of the female organs of generation and is a collective term for the labia minora, labia majora, mons veneris, clitoris, perineum, and vestibule of the vagina. Any or all of these parts may be involved in traumatic injury to the vulva, but the most common site is the labia majora. The labia majora are homologous to the scrotum in the male and are composed mainly of fatty tissue, but also contain the terminal insertions of the round ligaments. The main blood supply is the posterior labial artery which is a terminal branch of the internal pudendal. The clitoridal artery is also a terminal branch of the internal pudendal and its course is deep to the labia majora but may be ruptured with sufficient trauma. The fatty tissue of the labia majora is supplied with an

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extensive venous plexus which often contributes to the formation of a hematoma when traumatized.[3] In puerperal hematomata the vaginal and the azygous arteries of the vagina are frequently involved.

PATHOLOGY

Vulvar hematoma formation usually takes place within the labia majora. When trauma has been great and hemorrhage excessive there may be widespread extravasation and dissection upward along the natural cleavage lines of fascial layers (Table 1). Since the round ligaments insert into the labia majora, dissection of the hemorrhage may extend up the inguinal canal, into the broad ligament and retroperitoneally.

TABLE 1
AREA OF DISSECTION

	GYN.	OB.
1. To Anus	7	8
2. External inguinal ring	3	3
3. Internal inguinal ring	2	2
4. Vaginal vault	4	6
5. Suprapubically	2	0
6. Peri-urethrally	3	5
TOTAL	21	24

ETIOLOGY

In the nonobstetrical variety the etiological factor is extremely variable, but is due to some type of trauma. In the present series the most common injury was a fall.

The puerperal type, however, may be due to trauma or to an inadequate episiotomy repair. It seems logical that the increased incidence of varicosities of the vagina and vulva associated with pregnancy may be a factor. One case in this series had a spontaneous rupture of a varix with no trauma during the prenatal period. This patient was treated surgically, by placing a deep figure of eight suture of absorbable material through the area. She had an uneventful vaginal delivery one month later. Burke[4] has recently reported a similar case occurring during the prenatal period and this patient also had an uneventful vaginal delivery five months later.

DIAGNOSIS

In the traumatic variety there is always a history of trauma and the patient usually presents herself shortly thereafter complaining of severe pain and swelling. Adequate examination is almost impossible without anesthesia. It is our feeling that complete and careful examination is essential in these cases with particular attention being paid to possible injury of the urethra, bladder, or rectum.

In the puerperal type the patient usually complains of more than normal postpartum perineal pain. Ordinarily this does not occur until the patient has been transferred from the delivery unit to her room, thus intermittent inspections of the perineum during the first few hours after delivery will aid in the early detection of hematomata formation. Again, anesthesia is usually necessary to determine the extent of hemorrhage.

All patients should have the basic laboratory studies of their hemoglobin, white blood cell count, bleeding and clotting times and a catheterized urine specimen.

CLASSIFICATION

Many classifications have been used in reporting vulvar hematomas, from measurement in centimeters to grading them as small, medium, or large. While it is of value to be as accurate as possible in the description of the extent of the lesion the actual size of the hematoma presenting at the introitus has no great bearing on prognosis. The degree of extravasation beyond the vulva is of much greater importance. Thus, a good pelvic examination becomes mandatory, especially in order to determine whether the dissection has been into the broad ligament and possibly retroperitoneal. If this does occur the treatment will be more radical and from a different approach. Eastman[3] states that the cases collected by Williams in 1904, of subperitoneal hematoma had a mortality rate of 56 per cent. Hamilton[5] reported a mortality rate of 8.3 per cent in the retroperitoneal type since the introduction of active surgical therapy. There is also the possibility of intraperi-

toneal rupture from the broad ligament and a generalized peritonitis if infection ensues.

In the present series the average age was 24.1 years and the average size 7 by 8 centimeters.

In the traumatic type 75 per cent were on the right and 25 per cent on the left. In the puerperal type 16 followed the use of a left mediolateral episiotomy and all were on the same side as the episiotomy. Of the ones occurring after a spontaneous delivery, 2 were on the right and 7 on the left. It would seem evident from the above that the hematomas occurring after an episiotomy resulted from an inadequate repair rather than trauma.

TREATMENT

A survey of the literature indicates the consensus is conservative management (bed rest, cold packs, and pressure) for small hematomas and surgical treatment of expanding or very large ones. It is our feeling that more liberal use of surgery, even in small hematomas, may yield somewhat better results. Nearly all lesions have a lacerated or abraded surface from trauma, and infection with abscess formation is always a possibility. Furthermore, it is not possible to predict which hemorrhages will stop with conservative treatment and which will continue to enlarge, necessitating surgical intervention later when the likelihood of infection is greater. Some may rupture spontaneously, though usually with incomplete drainage. From this series the length of time necessary for complete recovery was much longer when conservatism was used.

Of the 21 traumatic hematoma, 10 were treated conservatively and 11 surgically. Of the 24 puerperal type, 2 were treated conservatively and 22 surgically. Morbidity was measured by the length of hospital stay and the degree of febrile reaction (the temperature of 100.4 degrees was used as the basis of a febrile reaction). Of the injuries treated conservatively 71 per cent were febrile; whereas only 12 of the 33 treated surgically were febrile, or 36 per cent.

The length of hospital stay was reduced almost 50 per cent when surgery was employed. The average length of stay with conservative treatment was 9.4 days and with surgery 5.0 days.

The surgery used in all cases except the 1 prenatal case mentioned previously was incision and evacuation of all clots and complete obliteration of the large denuded, oozing area with figure of eight or matress sutures of absorbable material. Drainage was not employed in any case and there were no seroma, abscess formation, or recurrence of the hematoma. If large bleeding vessels were seen and could be ligated separately, this was done.

No definite opinion could be formed from this study regarding the efficacy of antibiotic therapy. It is the feeling of this department that chemotherapy should be employed when specifically indicated and not as a routine procedure. However, when it is used we prefer either a combination of penicillin and streptomycin or one of the broad spectrum agents due to the large number of gram negative organisms in this area.

Upon a two-week follow-up examination, 28 of the 33 cases treated surgically were described as completely healed and the remaining 5 were tender but with no induration present. However, 7 of the 12 cases treated conservatively were tender with induration and 5 were listed as tender.

PROGNOSIS

The prognosis in both traumatic and puerperal hematomas is excellent if prompt surgical treatment is undertaken. In this series there were no deaths, no retroperitoneal involvement, and no cases of postoperative dyspareunia. Lyons [6] has reported a case of dyspareunia after surgical drainage of a vulvar hematoma followed by packing the area and allowing secondary healing to take place.

SUMMARY

1. Both traumatic and puerperal hematomas are relatively common, extremely painful, and very disabling.

2. The size and course of dissection is

variable and of great import. Anesthesia is usually necessary for an accurate diagnosis.

3. If treated conservatively these hematomas can continue to enlarge, liquify, or undergo abscess formation.

4. No complications occurred from prompt surgical intervention and the morbidity and length of hospital stay were greatly reduced with this treatment.

5. The two week follow-up examinations revealed a more satisfactory result in the cases treated surgically.

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IN MEMORIAM

Rudolph Matas, M. D.

Dr. Rudolph Matas, oldest member of the Louisiana State Medical Society and its most distinguished surgeon, died in New Orleans at Touro Infirmary, on September 23, 1957.

His career was one of fame and achievement. Its accomplishments were made possible by a great mind, and fortunately they were acknowledged during his own lifetime, in such a fashion as has come to few men. His friends, his associates in organized medicine, and the advancement of medicine over the world, owe him a debt of gratitude. When he entered the field of medicine in 1877, the modern era had not begun. The Darwinian theory of evolution was being discussed with vitriolic comments. The germ theory of disease, so-called, was just worthy of a passing glance. When a lecturer appeared in what is now the Tulane School of Medicine, in 1877, to speak on the Listerian method and the germ theory of disease, only four out of the student body of some two hundred appeared to listen. Dr. Matas was one of the four. When he died with his age in the late nineties, he had seen the most productive phase of the science and art of medicine progress through its most fruitful sixty-five years. For along this path of progress it was he who directed many aspects of this advancement in medical knowledge. His biographers will point to many "firsts" in medical achievement, and particularly to the Matas operation of endoaneurismorrhaphy. This was acclaimed the greatest advance in blood vessel surgery in over two hundred years and was the beginning of the present extraordinary developments in that field.

He was America's most honored surgeon, with evidences of acclaim here and abroad. Dr. William J. Mayo called him "the most learned surgeon I have ever known." Among his distinguished awards was the world's highest for general surgery, the Henry Bigelow Medal of the Boston Surgical Society. The world's highest award for vascular surgery is named in his honor.

His services in the cause of medicine as a teacher, his inspiration to his associates and students, and his benefactions to the Tulane School of Medicine, match his achievements in surgery. He devoted a large part of his time and energy for forty-two years to teaching in the Medical School of Tulane University. He was Professor of Surgery and Head of the department from 1894 to 1927, and Emeritus in 1927 until his death. His bequests to the University in the form of his medical library and in the financial pro-

visions of his will were a model of generosity and forethought.

Dr. Matas's achievements and fame were not the result of being tossed into prominence by the waves of fate. The pinnacle upon which he stood rested on a firm pyramid with a broad base of energy, effort, and painstaking attention to detail, which was not allowed to obscure the vision of the goal to be attained. His capacity for work and his physical endurance were in themselves remarkable. As a student, his hours of study were many times those of his roommates; at eleven o'clock when the others would go to bed, he was accustomed to put the lamp on the floor, stretch out on a blanket, and study on until 4:00 or 5:00 in the morning. This was repeated night after night, and at 7:00 A.M. he would be as fresh as any who had had the usual eight hours.

As a surgeon, he would repeatedly tell internes and residents to call him specifically at 1:00 or 4:00 A.M. to report progress of a seriously ill patient and to receive further instructions as to treatment.

As an author, he would rewrite and revise a manuscript repeatedly, giving endless care and thought to each sentence.

His fund of information and his cultural knowledge were a source of astonishment to all who had the privilege of contact with him. His choice of words in English was superb. He spoke fluent French, Spanish, Italian, Portuguese, good German and Romanian, and some portion of Scandinavian. In 1938, he addressed the delegates of the International Surgical Congress in four languages.

His services to organized medicine were distinguished and appreciated. He served on many committees in the interests of the Orleans Parish and the Louisiana State Medical Societies. He was President of the Louisiana State Medical Society and Vice President of The American Medical Association on two occasions and first recipient of its distinguished service medal (1938).

His generous bequest of five thousand dollars to the Orleans Parish Medical Society will benefit the present members and future generations.

He was Editor in Chief of this Journal for two years, 1883 to 1885, and Co-Editor, 1885 to 1890. From his bibliography of six hundred or more titles many were contributed to the pages of this Journal.

Dr. Matas attributed what he called his good fortune to inheriting good health and preserving his enthusiasm for things medical. To this must be added a rare capacity for sustained work directed by one of the world's great minds. With him went the satisfaction of having left the world better than he found it, and having advanced the cause of medicine by the vastness of his contributions. His career was and will be a living inspiration.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

DUES FOR 1958 MEMBERSHIP

Dues for 1958 membership in the Louisiana State Medical Society, American Medical Association and component parish and district societies, are payable now. The Parish and State Society dues may be included in one check and sent to the Secretary of your Parish Society. A separate check should be issued to the American Medical Association and should also be forwarded to the Secretary of the Parish Society, and he will forward State and AMA dues to the Secretary-Treasurer of the State Society. Dues for District Society should be sent to the Secretary of your district society.

Amount of dues is as follows: Louisiana State Medical Society, $50.00; American Medical Asso-

ciation, $25.00. Information in regard to amount of Parish and District Society dues should be secured from the Secretary of the Parish and District Societies.

DEATHS 1957
MEMBERS OF LOUISIANA STATE MEDICAL SOCIETY

Dr. Ernest Emile Allgeyer, Waveland, Mississippi, September 27
Dr. William Edward Barker, Jr., Plaquemine, October 5
Dr. Floyd Nash Beckcom, DeRidder, March 25
Dr. William Henry Block, Jackson, Mississippi, August 28
Dr. Ivy Devine Boyett, Kentwood, February 4
Dr. Houston Carlisle Chambers, Rayville, March 31
Dr. Frank Chetta, New Orleans, October 30
Dr. John David Frazar, DeRidder, March 9
Dr. Earl Jones, Alexandria, October 7
Dr. Joseph Starnes Kopfler, Kenner, January 16
Dr. Richard Shaffer Kramer, Jennings, February 10
Dr. Adolphe Landry, Delcambre, October 9
Dr. Burl Benjamin Lane, Jr., Zachary, September 17
Dr. Lewis Harris Levy, New Orleans, September 13
Dr. Rudolph Matas, New Orleans, September 23
Dr. Anees Mogabgab, New Orleans, April 18
Dr. Louis F. Robinson, Winnsboro, January 19
Dr. Robert Elmore Rowland, Pineville, March 11
Dr. Kirby Arthur Roy, Mansura, March 14
Dr. Leonard Case Scott, New Orleans, March 12
Dr. John B. Sutton, Shreveport, March 8
Dr. Samuel D. Yongue, Breaux Bridge, September 29

ACCIDENTS IN THE UNITED STATES *

Last year 95,000 Americans died prematurely because of injuries sustained in accidents. The annual number of accidental deaths averaged about 75,000 early in this century, but just before 1930 the yearly average rose to about 100,000 and has remained at that level.

The stability of the over-all accident death toll masks a considerable improvement in many aspects of the accident problem. Death rates per 100,000 population have declined by over 40 per cent, from 99.5, the peak in 1906, to 56.5 in 1956. For all accidents except motor-vehicle, the decline has been even sharper—more than two-thirds of the peak figure. The 1956 rate was 30.8 per 100,000.

In recent years accidents have consistently ranked fourth among the leading causes of death. They have been outranked only by such major illnesses as heart disease, cancer, and cerebrovascular lesions (mainly cerebral hemorrhage). The rise of accidents among the leading causes of death in this century has resulted from the declines in mortality from the major communicable diseases—not from any increase in accident mortality itself.

In 1956 accidents caused 6 per cent of all deaths in the United States, and at ages 1 to 36 they were the leading cause of death. Rates were highest at the upper ages, but accidents were heavily outranked as a cause of death at these ages by heart and other diseases associated with aging. Accidental death rates were nearly 2½ times higher among males than females, and higher among nonwhites than whites.

The costs of accidents are not felt merely in the loss of human lives. In 1956, according to the National Safety Council, accidental injuries numbered over 9½ million, including 350,000 resulting in some degree of permanent impairment. In dollars, total accident costs (including property damage, wage losses because of inability to work, etc.) were estimated at about $11.2 billion—almost as much as Americans spend for all private medical services.

Accidents occur in four kinds of environments —on the job, in public places, at home, and in motor vehicles. In the last 30 years, accidents on the job and in public places have decreased substantially, but those involving motor vehicles have increased as the volume of motor traffic has expanded.

On-the-job accidents

One of the brightest elements in the entire situation has been the success achieved against fatal accidents on the job ("work" accidents). In spite of a greatly expanded labor force, the death toll of workers on the job has been reduced from 19,000 in 1928—the first year these data became available—to 14,300, as estimated by the National Safety Council for 1956. Death rates in 1956, 23 per 100,000 workers, established a record minimum at just over half the peak rate of 43 in 1937.

The most hazardous industries in 1956 were mines and other extractive industries, where the mortality rate reached 100 per 100,000 workers. The construction industry followed with a rate nearly three-fourths as high, 71 deaths per 100,000 workers. The safest industries were trade, manufacturing, public utilities, and services with mortality below the national average.

Much of the success against work accidents has been due to intensive safety campaigns, resulting in increased safety consciousness among both employers and employees, and to improvements in conditions at work and in levels of living and of medical care. Protective devices have been adopted against the major accident and health hazards

* *Health Information Foundation* Vol. VI, No. 8, October, 1957.

in industry, including high temperature, silica dust, radiation, and toxic chemicals. Shorter working hours have helped to cut industrial fatigue, so often a factor in accidents. In many industrial processes the replacement of noxious substances by harmless ones, and the substitution of automatic machinery with proper safety devices for hazardous manual techniques, have helped considerably to reduce the accident toll.

Recreation, public transportation

Also encouraging is the substantial reduction in mortality from public accidents, i.e., non-motor-vehicle accidents occurring in public places. Moreover, this reduction has taken place in spite of the increased amount of leisure time enjoyed by the American people in recent years, with much of it devoted to sports and other recreational activities in public places. Deaths from these accidents numbered about 21,000 in 1928 but dropped to 16,000 by 1956.

About a fourth of these deaths—4,000 in 1956—were due to the drowning of persons while swimming or playing in water or falling into water. If drownings in transportation or boat accidents are included, the total exceeds 5,000. Falls and firearms combined accounted for an additional fourth of the public-accident death toll in 1956.

Almost an additional fourth—3,650 in 1956—involved some form of transportation, primarily air, water, and railroad. The largest toll—1,300 in 1956—came from aircraft accidents, both military and civilian. In view of the huge increase over the last few years in the volume of air traffic, this figure, although high, is still a considerable improvement over former years.

The largest number of fatal aviation accidents usually occurs in private flying, chiefly for pleasure and other noncommercial purposes. Domestic scheduled airlines have a superior record. In no year since the 1933-37 period has the death toll on scheduled airlines exceeded 200, and in only four years—1947, 1951, 1955, and 1956—has it exceeded even 100. Over the last 20 years the average annual death rate per 100 million passenger miles on these carriers was cut by over 90 per cent, from 7.80 in 1933-37 to 0.62 in 1956.

Just as heartening a story can be told about progress in railroad safety. Of the major forms of transportation, railroad passenger trains currently provide one of the safest means of passage, 0.20 deaths per 100 million passenger miles. Deaths in all types of railway accidents (not merely those directly involving the transportation of passengers) have declined sharply, from about 10,000 in 1918 to 2,600 in 1956. Since 1918, deaths of passengers have declined by more than 85 per cent, from over 500 in 1918 to 62 in 1956.

Accidents in the home

In the home the record has been less promising. With minor fluctuations, the number of deaths from home accidents has averaged around 30,000 each year since 1928; in 1956 the total was 28,000. Thus the American home was involved in nearly 30 per cent of all accidental deaths in 1956.

About half — 13,600 in 1956 — resulted from falls, primarily to persons aged 6 or over. Older people are especially vulnerable because of the greater amount of time they spend in the home and their frequently impaired physical condition.

Fatal burns from fires, and other deaths associated with fire, took over 5,000 lives in the home during 1956. Fire accidents are an important cause of death at all ages and are the leading cause of accidental death in the home at ages 1 through 64. Aged persons and children under five have the highest mortality rates and constitute half the victims in this type of accident.

A large proportion of the victims are trapped in homes destroyed by fire. Important among causes of death by burns in the home are careless smoking, accidental igniting of clothing on a stove or open fireplace, and explosion of cooking or heating appliances. In the past few years deaths have declined considerably as a result of general modernization of homes and widespread use of improved facilities for cooking, heating, lighting, and laundering.

About 850 deaths in the home were reported in 1956 as due to poisonous gas. Poisonings by solid or liquid substances accounted for an additional 1,050 deaths.

Motor-vehicle accidents

Accidents involving motor vehicles took over 40,000 lives in 1956, the largest number ever recorded. Deaths rose steadily early in the century from a low start at 400, the annual average for 1903-07, and by 1937 and 1942 peaks of just under 40,000 deaths were reached. Temporary declines during World War II were reversed by a sharp rise afterward. Except for a minor dip during 1954, the course of the death toll since 1950 has been steadily upward.

The rising number of deaths alone, however, tells only part of the story. Since 1900 the U. S. population has more than doubled, and so the death rate per 100,000 persons is a better measure of risk. This rate, like the number of deaths, reached peaks in 1931, 1937, and 1941 (28.3, 31.2, and 29.8, respectively). After declines during World War II, the rate climbed slowly, reaching 25.7 in 1956—still under the former high levels.

The recent increases in the population death rate are not surprising, in view of the growing importance of motor vehicles in American life. While just over 4,000 motor vehicles were produced in this country during 1900, over 9 million were turned out in 1955 alone. In 1956,

64.5 million motor vehicles were registered, while about 77 million drivers were licensed. Moreover, mileage traveled has increased at an almost phenomenal rate. Preliminary estimates indicate that motor vehicles traveled 630 billion miles in 1956, an average of just under 10,000 miles per vehicle.

As a result, a more realistic measure of motor-vehicle risk is the death rate per 100 million vehicle miles. This rate declined consistently, from a peak of 16.7 in 1934, to a record low of 6.3 in 1954. There was a slight rise to 6.4 in 1955 and 1956. This index suggests that safety in motor-vehicle travel has actually increased substantially since early in the century.

The greatest achievement has been the reduction in pedestrian deaths since 1937. From 1927 to 1937 these deaths rose from just under 11,000 to 15,500 but decreased thereafter, especially during the war. By 1956 the number stood at just under 8,000. Pedestrian death rates are lowest at ages 15-44, somewhat higher at ages under 15, and highest at 45 and over. In this last age group, nearly 5,000 such deaths occurred in 1956 —three-fifths of the total.

Noncollision accidents

A rising trend of deaths in noncollision accidents has largely offset the decrease in the number of pedestrian fatalities. The annual death toll in noncollision accidents averaged just under 9,000 from 1927 thru 1941, dropped to about 6,000 during the war years, and since then has risen steadily and rapidly, reaching 14,650 in 1956.

Rising even more sharply has been the toll from collisions between motor vehicles. Deaths in 1956 —13,850—were four times as numerous as those in 1927. Here, as with fatal noncollision accidents, persons in the age group 15-24 were the chief victims, and an overwhelming proportion of the accidents occurred in rural areas.

A recent investigation into one aspect of the motor-vehicle accident problem, violations by drivers in fatal accidents, showed that nearly one-third were exceeding the speed limit, or a safe speed, at the time of the accident. In urban areas, violating right-of-way was the next most common condition, while in rural areas failure to keep to the right of the center line followed in importance. In 22 out of 100 fatal accidents, a driver or an adult pedestrian had been drinking; "driving while under the influence of alcohol" was a factor in 7 per cent of all fatal accidents.

The weather was rainy, snowy, or foggy in one out of six fatal accidents, and in nearly as many cases there was some obstruction to the driver's vision. In nearly one out of 12 fatal accidents an unsafe condition was reported in at least one of the vehicles involved, most often unsafe brakes. About one out of 14 drivers had a physical condition—most often they were asleep—that could have been a contributing factor in the accident. About the same proportion of pedestrians in fatal accidents had physical defects.

Over one-fourth of the drivers in fatal accidents were between 18 and 24, and nine out of ten were men. But no valid conclusions can be drawn from these data about the comparative safety records of the different sex and age groups, since the total mileage driven by each is unknown.

In an attempt to reduce the motor-vehicle death toll, automobile manufacturers have begun to build protective mechanisms into their product, such as safety belts, doors that remain closed under impact, flexible steering wheels, dashboards without protrusions and covered with shock-absorbing material, and similar devices. In addition, an important step was taken by the directors of the Automobile Manufacturers Association last June when they unanimously agreed to de-emphasize speed and horsepower in the industry's advertising. For such preliminary measures to be intensified, however, the public must be educated to accept and even to demand at least these minimum protections.

In general, safety against accidents has achieved the greatest success where society has been able to bring its organized influence to bear. Safety regulations are stringently enforced in industry, railroads, scheduled air transport, public beaches, and similar environments, with gratifying results.

On the other hand, where the individual himself must assume most of the responsibility for his own safety and in many instances for that of others (in such places as the home and motor vehicle) safety progress has been slower. Although accidents are often beyond human control, a large element of carelessness and irresponsibility is involved in others. Perhaps the only real solution to the accident problem lies in intensified educational activities designed to spread safety conditions among all parts of the population.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

OFFICERS OF THE SURGICAL ASSOCIATION OF LOUISIANA FOR THE YEAR 1958

The following officers were elected for the year 1958 at the tenth annual meeting, held at the St. Charles Hotel, New Orleans, November 3, 1957: Charles R. Walters, M. D., President; Gordon W. Peek, M. D., 1st Vice-President; Walter F. Becker, M. D., 2nd Vice-President; E. L. Leckert, M. D., Treasurer; Henry G. Butker, M. D., Secretary. Additional members of the Board of Directors and their term of office are: Isidore Cohn, M. D., 1957-60; T. Jeff McHugh, M. D., 1957-59; Daniel J. Fourrier, M. D., 1955-58; Richard L. Buck, M. D., 1956-59; W. Robyn Hardy, Sr., M. D., 1956-59; C. Grenes Cole, M. D., 1957-60; H. H. Hardy, M. D., 1957-58.

LOUISIANA CHAPTER AMERICAN COLLEGE OF CHEST PHYSICIANS

A meeting of the Louisiana Chapter of the American College of Chest Physicians will be held on Friday, December 6, 1957, at the Veterans Administration Hospital, New Orleans, from 2:00 p. m. to 4:30 p. m. The scientific program will consist of the following: "Physiological Manifestations of Alveolar Hypoventilation," by Alfred P. Fishman, M. D., New York, and "Current Aspects in the Surgical Treatment of Acquired Cardiac Diseases," by Charles A. Beskin, M. D., Baton Rouge.

An important business meeting will follow which will include election of officers and consideration of plans for the Louisiana Chapter for the coming year.

LOUISIANA STATE SOCIETY OF ANESTHESIOLOGISTS

At the annual business meeting on November 14, 1957, of the Louisiana State Society of Anesthesiologists, the following officers were elected: President, Richard H. Morris, M. D., Alexandria; Vice-President, John B. Parmley, M. D., New Orleans; Secretary-Treasurer, William E. Trotti, M. D., New Orleans; Delegate, Wallace Merriam, M. D., New Orleans; Alternate Delegate, Vernon C. Fagan, M. D., New Orleans.

SECTIONAL MEETING AMERICAN COLLEGE OF SURGEONS

Jackson, Mississippi, January 16-18, 1958

All members of the medical profession are invited to attend a three-day Sectional Meeting of the American College of Surgeons in Jackson, Mississippi, January 16-18, 1958, at the Hotel Heidelberg.

Dr. J. Harvey Johnston, Jr., Clinical Assistant Professor of Surgery, University of Mississippi School of Medicine, is Chairman of the Local Advisory Committee on Arrangements.

An innovation at this year's Sectional Meetings is the Fellowship Luncheon, featuring a panel discussion on College activities with a question period. The president of the College, Dr. William L. Estes, Jr., will preside.

Dr. Johnston will preside over the opening session Thursday morning, January 16. This session will include the following reports:

Choice of Operation in Diseases of the Colon. John M. Waugh, M. D., FACS, Rochester, Minnesota.

Management of Mediastinal Thyroids. George E. Twente, M. D., FACS, Jackson.

Treatment of Hand Injuries. Daniel C. Riordan, M. D., New Orleans.

Panel Discussion on Complications of Abdominal Surgery:

Moderator: James D. Hardy, M. D., FACS, Jackson.

Collaborators: John J. Farrel, M. D., FACS, Miami; Howard Mahorner, M. D., FACS, New Orleans; Harwell Wilson, M. D., FACS, Memphis; Robert M. Zollinger, M. D., FACS, Columbus.

Panel Discussion on Cancer: Chemotherapy, Metastasis and Limitations of Surgery:

Presiding: Willard H. Parsons, M.D., FACS, Vicksburg.

Moderator: I. S. Ravdin, M. D., FACS, Philadelphia, Chairman, Board of Regents, American College of Surgeons.

Collaborators: Alton Ochsner, M. D., FACS, New Orleans; Albert Segaloff, M. D., New Orleans; Robert D. Sloan, M. D., Jackson.

Dr. Curtis P. Artz, Jackson, will preside over the afternoon session:

Common Errors in Management of Fractures. Thomas H. Blake, M. D., FACS, Jackson.

Treatment of Thrombosis of Major Veins. Howard Mahorner, M. D., FACS, New Orleans.

Diagnosis and Treatment of Surface Tumors. J. Harold Conn, M. D., FACS, Jackson.

Symposium on Pancreas and Liver:

Leader: J. Harvey Johnston, Jr.

Acute Pancreatitis. J. Harvey Johnston, Jr.

Chronic Pancreatitis. Robert M. Zollinger, M. D., FACS, Columbus.

Results of Radical Resection for Carcinoma of the Pancreas and Ampulla of Vater. John M. Waugh, M. D., FACS, Rochester.

Recognition and Therapy of Acute Liver Failure. John R. Snavely, M. D., Jackson.

A dinner tour of Mississippi's new medical center will be held Thursday evening, with Dr. James D. Hardy presiding. Dr. I. S. Ravdin will preside over the motion picture program which follows from 8 to 10 p. m.

Dr. T. E. Ross, Jr., Hattiesburg, will preside over the Friday morning session:

Adrenalectomy in Breast Cancer. Willard H. Parsons, M. D., FACS, Vicksburg.

Diagnosis of Massive Gastrointestinal Bleeding. John J. Farrell, M. D., FACS, Miami.

Early Management of Burns. William H. Moretz, M. D., FACS, Augusta.

The Status of Antibiotic Prophylaxis. Fred Allison, Jr., M. D., Jackson.

Symposium on Management of Multiple Injuries:

Presiding: Jones W. Lamb, M. D., FACS, Greenwood.

Leader: John M. Howard, M. D., FACS, Emory University.

Principles of Wound Management. Champ Lyons, M. D., FACS, Birmingham.

Emergency Management of Fractures. Robert A. Knight, M. D., FACS, Memphis.

Treatment of Acute Head Injuries. Dean H. Echols, M. D., FACS, New Orleans.

Treatment of Acute Chest Injuries. Rudolf J. Noer, M. D., FACS, Louisville.

Urologic Injuries. James W. Headstream, M. D., FACS, Little Rock.

Dr. Watts R. Webb, M. D., FACS, Jackson, will preside over the luncheon panel discussion:

New Horizons in Cardiac and Lung Surgery:

Moderator: H. William Scott, Jr., M. D., FACS, Nashville.

Collaborators: Oscar Creech, Jr., M. D., FACS, New Orleans; Edward F. Parker, M. D., Charleston; Watts R. Webb.

Dr. George E. Twente, M. D., FACS, Jackson, presides over the P.M. session:

Symposium on Pediatric Surgery:

Pre- and Postoperative Care of Infants. James N. Etteldorf, M. D., Memphis.

Teratomas in Children. Hugh B. Lynn, M. D., FACS, Louisville.

Abdominal Emergencies in Infants. C. Everett Koop, M. D., FACS, Philadelphia.

Urinary Anomalies in Children. Temple Ainsworth, M. D., FACS, Jackson.

Panel Discussion on Nutrition Therapy and Transfusions in Surgical Patients:

Moderator: Curtis P. Artz, M. D., FACS, Jackson.

Collaborators: Warren N. Bell, M. D., Jackson; James N. Etteldorf, M. D., Memphis; Champ Lyons, M. D., FACS, Birmingham.

Hodding Carter, Owner and Publisher of the **Delta Democrat Times**, will be the dinner guest speaker Friday evening. Dr. J. Harvey Johnston, Jr., M. D., FACS, Jackson, will preside.

On Saturday, the final day of the meeting, Dr. Robert B. McLean, M. D., FACS, Jackson, will serve as presiding officer:

Present-Day Concepts in Treatment of Intestinal Obstruction. Rudolf J. Noer, M. D., FACS, Louisville.

Ovarian Tumors. Conrad G. Collins, M. D., FACS, New Orleans.

Urologic Complications in Abdominal Surgery. James W. Headstream, M. D., FACS, Little Rock.

Panel Discussion on Treatment of Carcinoma of the Cervix, Present Status:

Moderator: Conrad G. Collins, M. D., FACS, New Orleans.

Collaborators: Vincent P. Collins, M. D., Houston; Michael Newton, M. D., FACS, Jackson; Cyrus C. Erickson, M. D., Memphis.

The Sectional Meeting will close with the Fellowship Luncheon.

Dr. H. Prather Saunders, Associate Director, American College of Surgeons, is in charge of all Sectional Meetings for the College.

LOUISIANA STATE UROLOGICAL SOCIETY

The next meeting of the Louisiana State Urological Society will be held in Lafayette, Louisiana. Officers elected for this year are: President, Dr. Thomas Kimbrough; Vice-president, Dr. Thom-

as Latiolais; Secretary-Treasurer, Dr. William Vildebil.

The past meeting was held in January at Shreveport. Attending this week-end meeting were ninety-two Urologists of the states of Louisiana, Texas, Arkansas, Mississippi, and Oklahoma. Dr. Ormond Culp of Rochester, Minnesota and Dr. Robert McIver of Jacksonville, Florida were the guest speakers. Scientific Papers were also presented by Dr. Eugene Vickery of New Orleans, Dr. W. Kitteridge of New Orleans, and Dr. Murphy Yearwood of Shreveport.

An outstanding event of this meeting was the *Cajun Oyster Bust.* This was a stag affair and the Doctors opened their own oysters. The oysters were served in every eatable manner. This entertainment was the highlight of the whole week-end.

CONTAMINATED WATER MAY PLAY ROLE IN POLIO SPREAD

When an outbreak of polio occurs in a specific area within a community, it may be caused by pollution of the area's drinking water, three Nebraska researchers reported.

While a community's water may be judged safe at pumping stations and other regular testing sites, it may become contaminated within a small area of the distribution system. Therefore, the water in an outbreak area should be tested and, if necessary, boiled before drinking, they said in a recent (June 22) Journal of the American Medical Association.

Dr. Paul M. Bancroft, Warren E. Engelhard, Ph.D., and Dr. Charles A. Evans, Lincoln, Neb., studied an unusual polio outbreak occurring in the summer of 1952 in Huskerville, a community of University of Nebraska students and their families near Lincoln.

All but two cases, one paralytic and one non-paralytic, occurred in two and one-half of the four rows of barracks-like buildings. During a five-week period, more than 10 per cent of the 347 children in the affected two and one-half rows developed poliomyelitis and 4.6 per cent suffered paralytic polio. During this same period, there were no cases among the 256 children residing in the adjoining section of the village.

The persons living in the village were "strikingly select," in that they were of approximately the same age and of the same cultural, social and physical backgrounds. There were no geographical or social barriers and no biological or other environmental features that accounted for the unusual distribution of cases. Measles and chickenpox spread through the same community without any higher incidence occurring in particular areas, the authors said.

A variety of evidence pointed to pollution of the water supply within the affected area as "the sole factor" which could be used to explain the disease distribution, they said.

Contaminated water has long been suggested as a mode of spreading polio; however, there has been no convincing evidence presented that pollution of a community water supply was responsible.

In the Huskerville outbreak, it appears that it was not the community water system, but only water near the affected individuals that wes contaminated. If this is true, it means that the role of water in the spread of polio must be re-examined, the authors said.

Institutional epidemics or the clustering of cases in certain buildings or even in certain parts of buildings, or in certain small villages, might be explained if pollution of water near individuals is an important factor, they said.

Until the significance of "proximate pollution" of water in the spread of polio can be evaluated, the boiling of drinking water in the home is a measure that should be considered as one precaution of possible value in prevention of the disease in times of epidemic, especially when it is remembered that cross connections and other conditions favoring localized contamination of water supplies are still common in many water systems, they concluded.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

ORLEANS PARISH

The Woman's Auxiliary to the Orleans Parish Medical Society entertained at a program tea Wednesday afternoon, November 11th, at the Orleans Club on St. Charles Avenue.

The president, Mrs. Eugene H. Countiss, presided at the meeting.

Mrs. Charles Farris, Jr., program chairman introduced Mr. Fred Wagner who gave a most interesting talk on one of his hobbies, "Making your own Christmas Cards."

Receiving the members and their guests with Mrs. Countiss were four of the board members: Mmes. Nicholas Chetta, chairman of Essay Contest; George F. Sustendal, chairman Doctor's Day; Spencer B. McNair, chairman Nurse Recruitment; and Edwin A. Socola, chairman Public Relations.

Mrs. Fred O. Brumfield, chairman of hostesses, was assisted in the dining room by Mmes. Shelley R. Gaines, Clarence E. Black, Jr., Vincent P. Blandino, Edward R. Christian, Joseph J. Ciolino, Anthony Failla and Carlo P. Cabibi.

The tea table centered with a lovely arrangement of pink roses was presided over by Mrs. Robyn Hardy and Mrs. L. Sidney Charbonnet, Jr.

Mrs. Branch J. Aymond,
Publicity Chairman

BOOK REVIEWS

The Physician-Writer's Book . . . Tricks of the Trade of Medical Writing: by Richard M. Hewitt, A.M., M.D. Philadelphia, W. B. Saunders Company, 1957. pp. 415. Price $9.00.

For a great many years Dr. Richard M. Hewitt has supervised the publications of the Mayo Clinic. He is therefore admirably fitted for the task of teaching the tricks of medical writing to both authors and editors. Not many of them are omitted in the seven sections which comprise his book on the subject. There is also a final section consisting of twenty-one appendices, which this reviewer would have preferred to have follow the particular chapters to which they are applicable.

This is a book to dip into rather than to read consecutively. Its usefulness is greatly increased by an index of twenty-one pages from which, an extensive sampling shows, nothing important has been omitted.

Perhaps the best way to convey the field and value of this book is to comment upon special items:

When, says Dr. Hewitt, the editor's foibles or the writer's desires come into conflict with the publisher's policies, it is the publisher who has the last word. For that reason, among others, the writer should not trouble himself too much with such matters as hyphenation.

The author's name should always be signed in exactly the same way, to which it might be added, particularly if his is one of the more common names.

A great deal of attention should be paid to the wording of the title and the subtitle, the latter being one way of simplifying life for both indexers of the literature and searchers thereof. A word might have been said on the wisdom—at least when the makers of programs permit—of formulating the title after the contribution is prepared, since the latter does not always turn out as originally planned.

Illustrations are expensive. They should therefore be carefully prepared and not employed at all unless they tell the story better than the text. Due regard should be paid to the quality of the paper used in the journal in which they are to be published; if the paper is of inferior grade, it might be best to eliminate the illustrations.

Tables presenting similar data should be prepared in similar fashion and their headings should be parallel. The sound distinction is made between the presentation of numerical data and their statistical analysis. Errors in numerical data are made with the greatest of ease; the not entirely humorous anecdote is told of two very capable physicians who had seized every possible chance in their paper to make the text and tables disagree.

The same point of view should be maintained throughout a paper. Tenses should be used with consistency. The reference of pronouns should be correct, and the widely used "this" must have proper ancestry. Listings should be in the same form throughout, not a combination of nouns and sentences and clauses.

Case is not a synonym for patient. It is too bad that the unspeakable "case where" construction was not castigated.

Abbreviations should be used with caution and preferably not at all. The human memory is surprisingly frail.

Transitional words are of great value. The limitations of the language make the list of such words brief and it is unfortunate that the very useful nonetheless was omitted from it.

Note-taking is basic but useful only when there is one idea to a note. No one who has not followed this simple plan has any idea of how helpful it can be.

In some of the usages he advises—or to which he takes exception—Dr. Hewitt has already lost his battle. Webster sanctions the plural of such words as exostosis and metastasis, and this usage seems simpler and more direct than exostotic lesions and metastatic growths. Webster is also willing for the physician to transfuse the patient and so is this reviewer. Colostomy may be less desirable than colonic stoma, but there is a medical metonymy and such terms as "functioning colostomy" have apparently come to stay. It is too bad that Dr. Hewitt did not pay his respects to the pretentious word anesthesiologist; the simpler term anesthetist was apparently a total casualty of the war years.

On the whole, however, Dr. Hewitt's policies and practices are entirely commendable and very sensible. He warns, for instance, against the promiscuous use of distal and proximal and calls for the judicious use of former and latter. He speaks sensibly about what he calls strong repetition, which is deliberate and intentional and is achieved by parallelism. He also mentions the achievement of emphasis by the position of a thought, whether in the sentence or the paragraph, in neither of which is a central location the place to accomplish it.

Dr. Hewitt makes the distinction between inclusive and selective reviews of the literature and says some wise words about omitting both when neither is needed. More emphasis might have been put upon selectivity in preparing to write a paper; reading everything on the subject can be both wasteful of effort and extremely confusing. He apparently approves the practice, to which this reviewer takes exception, of omitting titles to save space in a list of references and of tying the text into center and side headings for the same purpose. Diffuseness is, of course, to be avoided, but

the impression is given, perhaps unintentionally, that it is always desirable to be brief. Long sentences, if properly constructed, can be both clear and forceful, and too much condensation may be the road to obscurity, particularly if the writer, in the fulness of his own knowledge, omits some of the steps which the reader may need.

This reviewer cannot agree with Dr. Hewitt in the methods suggested for what he calls "the rearrangement" of the article. A paper which needs what was done to his example would be best handled by complete rewriting, if only by the criterion of the aphorism that there in no good writing, only good re-writing. When rearrangement of material is necessary in the final copy, enclosing the sentences (or clauses or phrases or words) in parentheses and numbering them in the desired order will do the job much more efficiently than Dr. Hewitt's cut-and-paste technique; the method suggested has been found, by experience, to be practically fool-proof, even to inexperienced typists.

It is often wise advice to destroy what one has written, particularly, as Dr. Samuel Johnson said, if the passage seems particularly fine to its creator. Quiller Couch said it more directly—hold the manuscript for a certain time, read it over, and then "murder your darlings."

Dr. Hewitt says in his preface that he has been accused of being too lavish in his credits to others. The accusation is well founded. It is delightful, however, to find a physician who can quote, with equal facility, from Dickens, Cervantes and Ian Maclaren and from Gowers and Quiller Couch and Fowler. He seems to have missed only one very useful reference work, the *Oxford Authors' and Printers' Dictionary.* In another edition he will undoubtedly quote from Margaret Nicholson's excellent adaptation of Fowler's classic text; Miss Nicholson's book was published after Dr. Hewitt's had appeared.

Dr. Hewitt, as this review indicates, has produced a useful book. This reviewer would have classified it as such if she had found in it no more than these two ideas, that the attempt to misunderstand a manuscript is the basis of the editorial function and that it is an indexer's duty to make an item discoverable under any heading beneath which a reasonable person would look for it.

ELIZABETH M. MCFETRIDGE, M.A.

Services for Children with Hearing Impairment; by American Public Health Assoc., Inc., N. Y. 1956, pp 124, (no price noted).

This booklet carries the subtitle, *A Guide for public health personnel.* Its usefulness will extend over a much wider field. It is one of a series which "is directed chiefly to persons in voluntary or official agencies or community organizations whose decisions singly or jointly determine the extent, coverage, content and operation of community services to children, particularly those who are handicapped.

It presents a very thorough, comprehensive coverage of the problems of the hearing handicapped child. The material is well organized and not too technical for one not well acquainted with the field. For the specialist in this area there are the Appendices in which much worthwhile factual information is gathered together.

Otologists might take exception to the recommendation of antibiotic therapy for acute otitis media without any further qualifying statement. It must be remembered that this is a general presentation of the total aspects of hearing-loss and as such merely mentions some of the controversial questions.

It is highly recommended for any one interested in hearing impairments whether from an academic or personal aspect.

JEANNETTE K. LAGUAITE, PH.D.

PUBLICATIONS RECEIVED

The C. V. Mosby Co., St. Louis: Allergy in Pediatric Practice, by William B. Sherman, M. D., and Walter R. Kessler, M. D.

Philosophical Library, Inc., N. Y.: Fear: Contagion and Conquest, by James Clark Moloney, M. D.

W. B. Saunders Co., Phila.: Introduction to Anesthesia, The Principles of Safe Practice, by Robert D. Dripps, M. D., James E. Eckenhoff, M. D., and Leroy D. Vandam, M. D.

Charles C Thomas, Publisher, Springfield, Ill.: A System of Ophthalmic Illustration, by Peter Hansell, M.R.C.S.; Ear, Nose and Throat Dysfunctions due to Deficiencies and Imbalances, by Sam E. Roberts, M. D., with a foreword by Morris Fishbein, M. D.; Digitalis, compiled and edited by E. Grey Dimond, M. D.; Host-Parasite Relationships in Living Cells, a Symposium sponsored by The James W. McLaughlin Fellowship Program, University of Texas, Medical Branch; The Physician's Own Library, by Mary Louise Marshall; Psychobiology, a Science of Man, by Adolph Meyer, M. D.; Introduction to Clinical Endocrinology, by A. Stuart Mason, M. D.; The Epileptic Seizure, by Cosimo Ajmone-Marsan, M. D., and Bruce L. Ralston, M. D.; Clinical Pathology Data, by C. J. Dickinson, B.A., with foreword by C. E. Dent, M. D. (2nd edit.); Bone Tumors, by David C. Dahlin, M. D.; Roentgen Diagnosis of Abdominal Tumors in Childhood, by Charles M. Nice, Jr. M. D., and Alexander R. Margulis, M. D., and Leo G. Rigler, M. D.; Anatomies of Pain, by K. D. Keele, M. D.; Tuberculosis, by J. Arthur Myers, M. D.; Recovery From Schizophrenia, by John Eisele Davis, Sc.D.

The Williams & Wilkins Co., Balt.: May's Manual of Diseases of the Eye, revised and edited by Charles A. Perera, M. D. (22nd edit.)

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